NEUR 1P90
Library Seminar
Fall 2023 term

Ian Gordon

Ian Gordon, Teaching & Learning Librarian

Brock University Library

Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers

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Top 10 resources you should know about!

1. Zoterobib
2. Zotero
3. Omni
4. Borrowing from Other Libraries
5. Brock Library NEUR Library Research Guide
6. Google Scholar, Google Books and Advanced Google searching
7. Open Education Resources (OERs)
8. PubMed / MEDLINE
9. When, where and how to get help!
10. Book a Consultation

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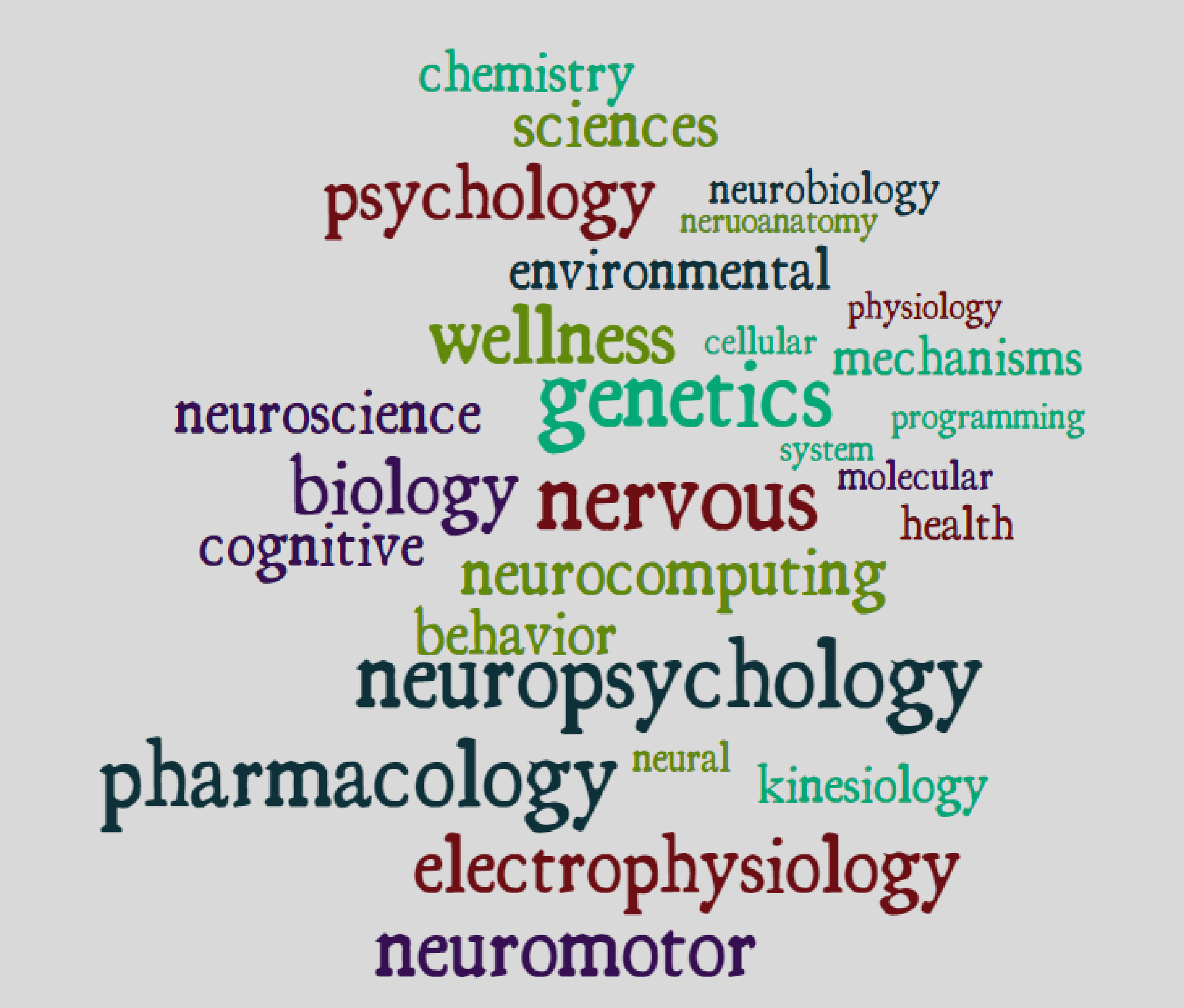
What is neuroscience?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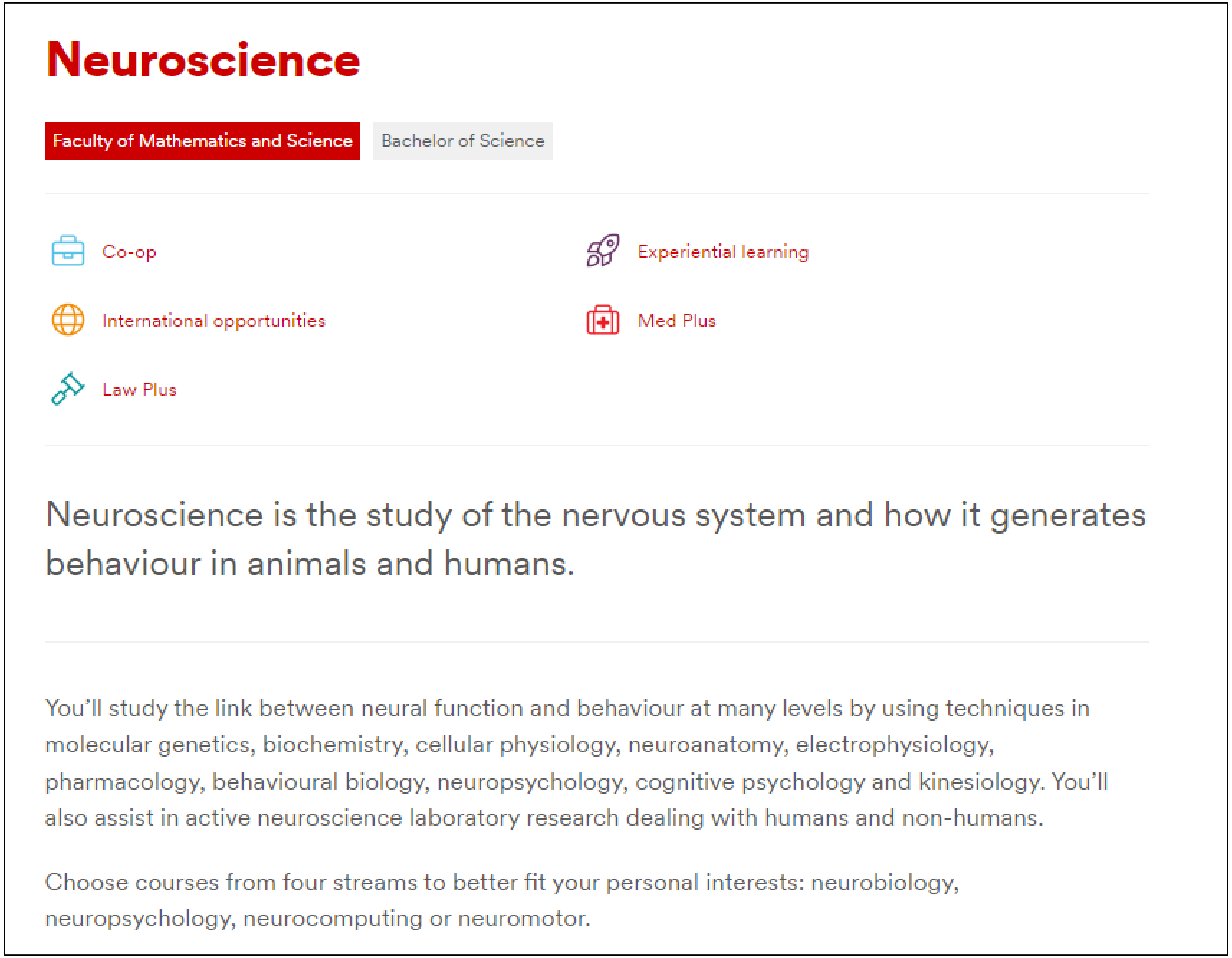

Neuroscience

Faculty of Mathematics and Science

Bachelor of Science

- Co-op
- Experiential learning
- International opportunities
- Med Plus
- Law Plus

Neuroscience is the study of the nervous system and how it generates behaviour in animals and humans.

You'll study the link between neural function and behaviour at many levels by using techniques in molecular genetics, biochemistry, cellular physiology, neuroanatomy, electrophysiology, pharmacology, behavioural biology, neuropsychology, cognitive psychology and kinesiology. You'll also assist in active neuroscience laboratory research dealing with humans and non-humans.

Choose courses from four streams to better fit your personal interests: neurobiology, neuropsychology, neurocomputing or neuromotor.

Let’s find a definition we can use in an essay, lab, seminar…

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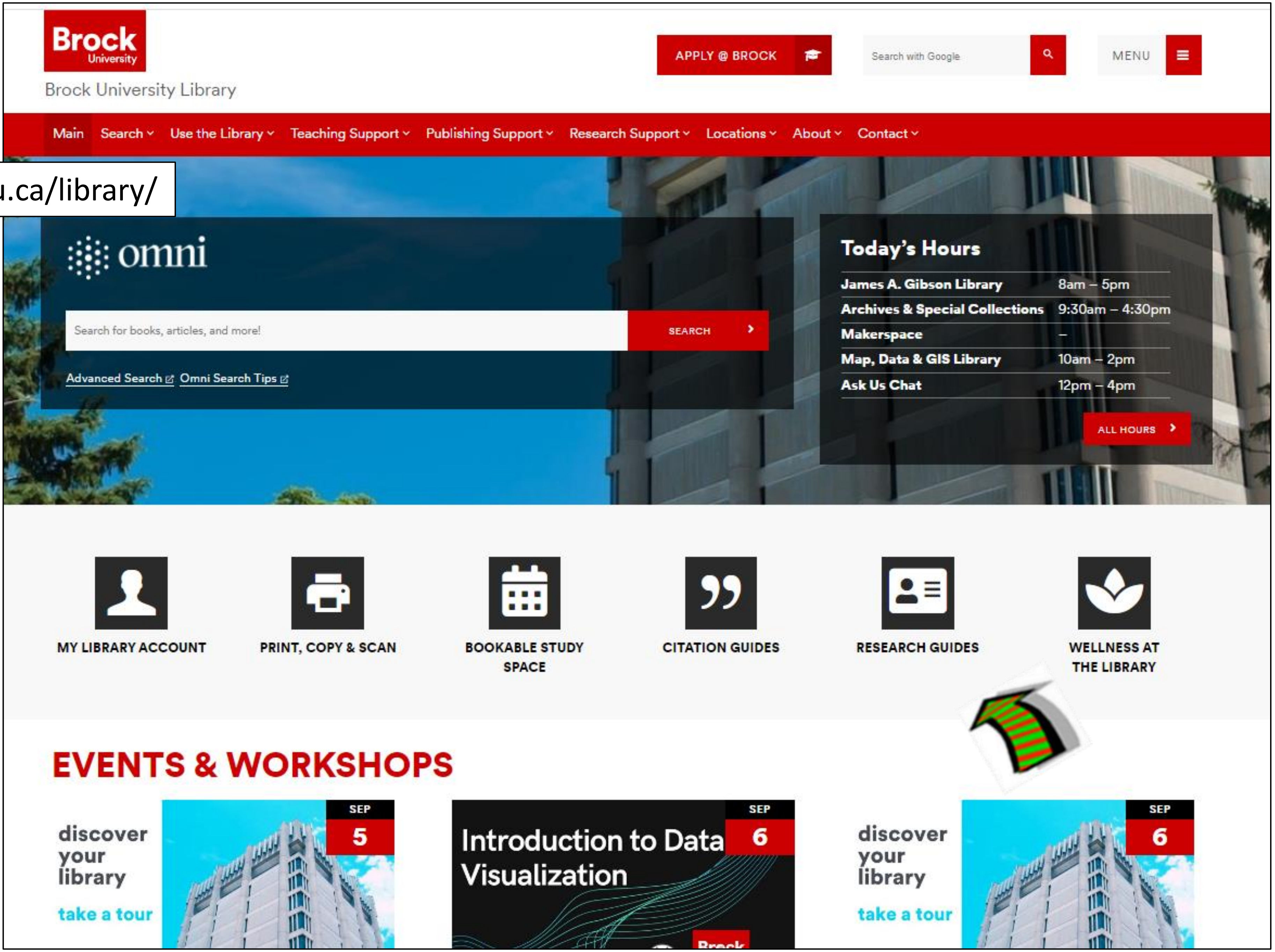

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Today's Hours

James A. Gibson Library	8am – 5pm
Archives & Special Collections	9:30am – 4:30pm
Makerspace	–
Map, Data & GIS Library	10am – 2pm
Ask Us Chat	12pm – 4pm

EVENTS & WORKSHOPS

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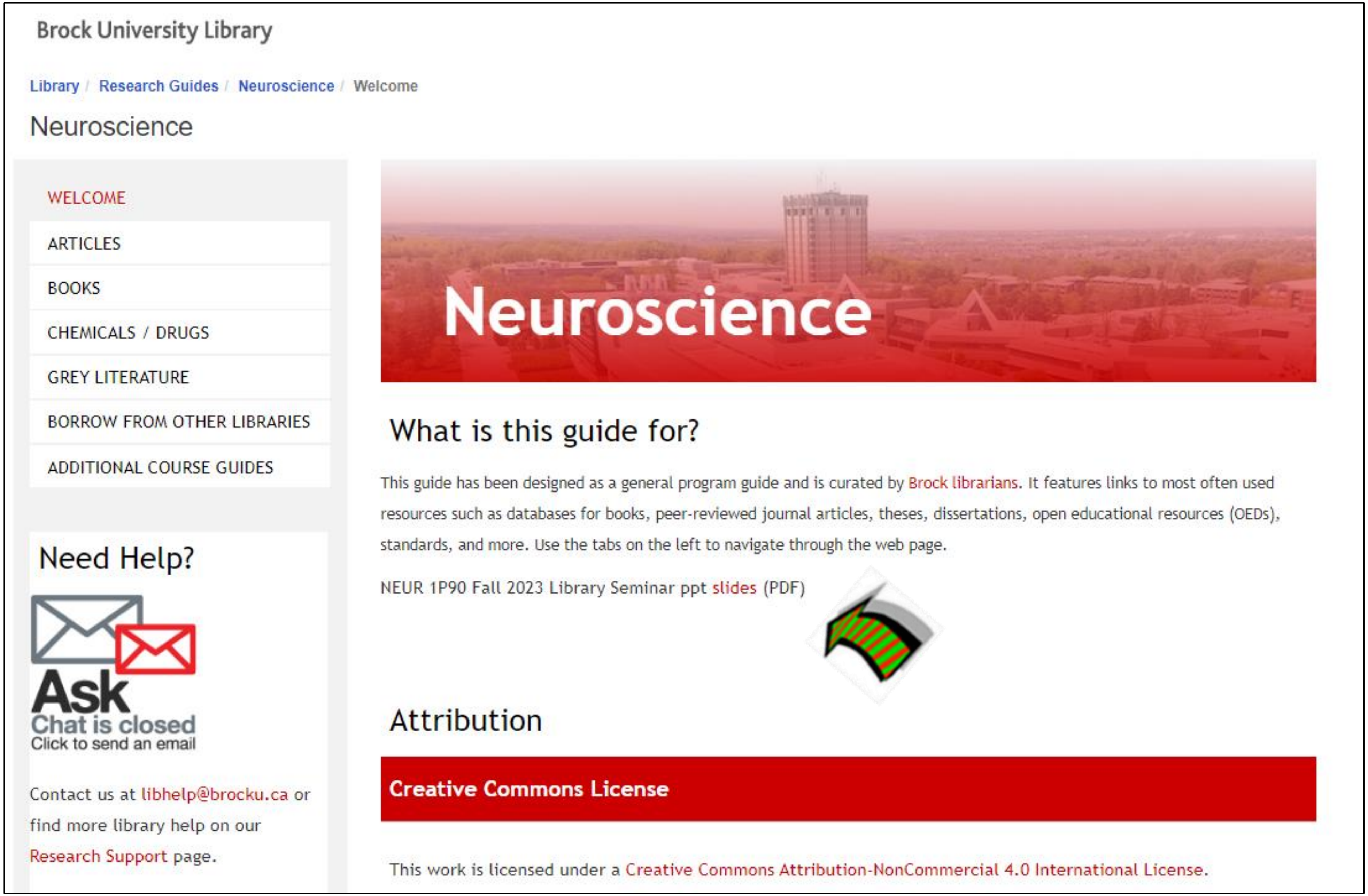
SEP 6 — Introduction to Data Visualization

SEP 6 — discover your library take a tour



Neuroscience

What is this guide for?

This guide has been designed as a general program guide and is curated by Brock librarians. It features links to most often used resources such as databases for books, peer-reviewed journal articles, theses, dissertations, open educational resources (OEDs), standards, and more. Use the tabs on the left to navigate through the web page.

NEUR 1P90 Fall 2023 Library Seminar ppt slides (PDF)

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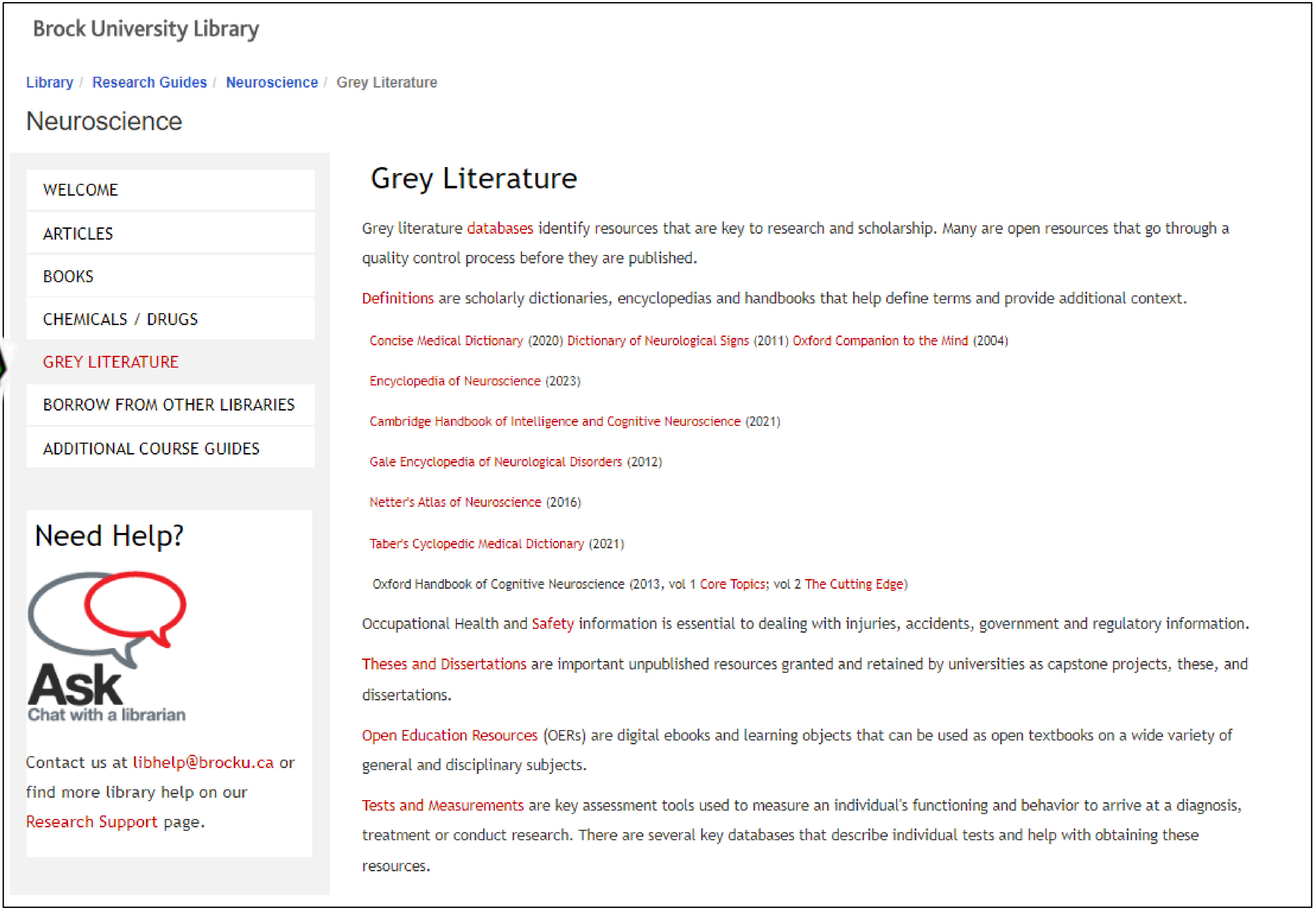

Neuroscience

Grey Literature

Grey literature databases identify resources that are key to research and scholarship. Many are open resources that go through a quality control process before they are published.

Definitions are scholarly dictionaries, encyclopedias and handbooks that help define terms and provide additional context.

Concise Medical Dictionary (2020) Dictionary of Neurological Signs (2011) Oxford Companion to the Mind (2004)

Encyclopedia of Neuroscience (2023)

Cambridge Handbook of Intelligence and Cognitive Neuroscience (2021)

Gale Encyclopedia of Neurological Disorders (2012)

Netter's Atlas of Neuroscience (2016)

Taber's Cyclopedic Medical Dictionary (2021)

Oxford Handbook of Cognitive Neuroscience (2013, vol 1 Core Topics; vol 2 The Cutting Edge)

Occupational Health and Safety information is essential to dealing with injuries, accidents, government and regulatory information.

Theses and Dissertations are important unpublished resources granted and retained by universities as capstone projects, these, and dissertations.

Open Education Resources (OERs) are digital ebooks and learning objects that can be used as open textbooks on a wide variety of general and disciplinary subjects.

Tests and Measurements are key assessment tools used to measure an individual's functioning and behavior to arrive at a diagnosis, treatment or conduct research. There are several key databases that describe individual tests and help with obtaining these resources.

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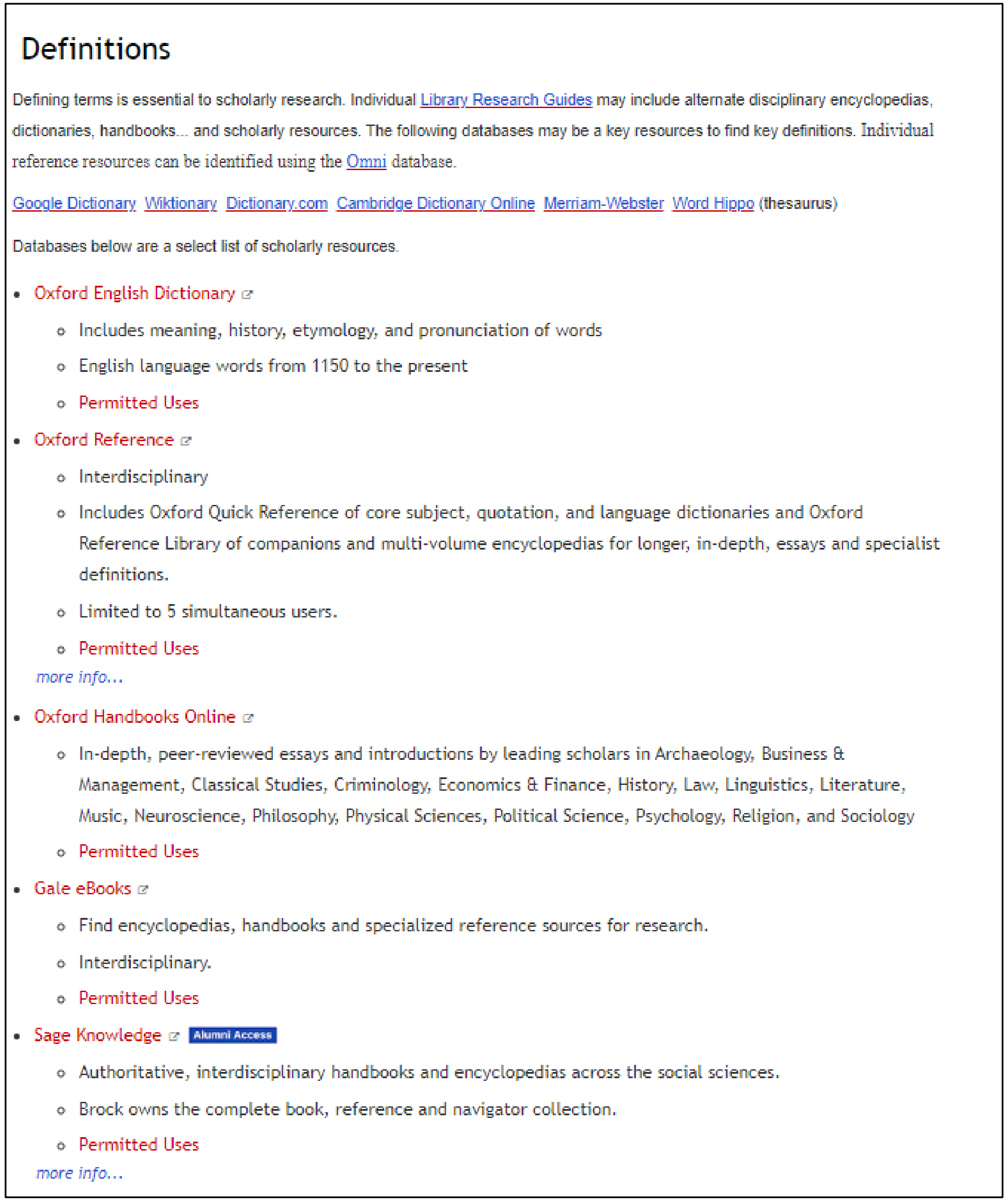

Definitions

Defining terms is essential to scholarly research. Individual Library Research Guides may include alternate disciplinary encyclopedias, dictionaries, handbooks... and scholarly resources. The following databases may be a key resources to find key definitions. Individual reference resources can be identified using the Omni database.

Google Dictionary Wiktionary Dictionary.com Cambridge Dictionary Online Merriam-Webster Word Hippo (thesaurus)

Databases below are a select list of scholarly resources.

- Oxford English Dictionary
  - Includes meaning, history, etymology, and pronunciation of words
  - English language words from 1150 to the present
  - Permitted Uses
- Oxford Reference
  - Interdisciplinary
  - Includes Oxford Quick Reference of core subject, quotation, and language dictionaries and Oxford Reference Library of companions and multi-volume encyclopedias for longer, in-depth, essays and specialist definitions.
  - Limited to 5 simultaneous users.
  - Permitted Uses

  more info...
- Oxford Handbooks Online
  - In-depth, peer-reviewed essays and introductions by leading scholars in Archaeology, Business & Management, Classical Studies, Criminology, Economics & Finance, History, Law, Linguistics, Literature, Music, Neuroscience, Philosophy, Physical Sciences, Political Science, Psychology, Religion, and Sociology
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  - Interdisciplinary.
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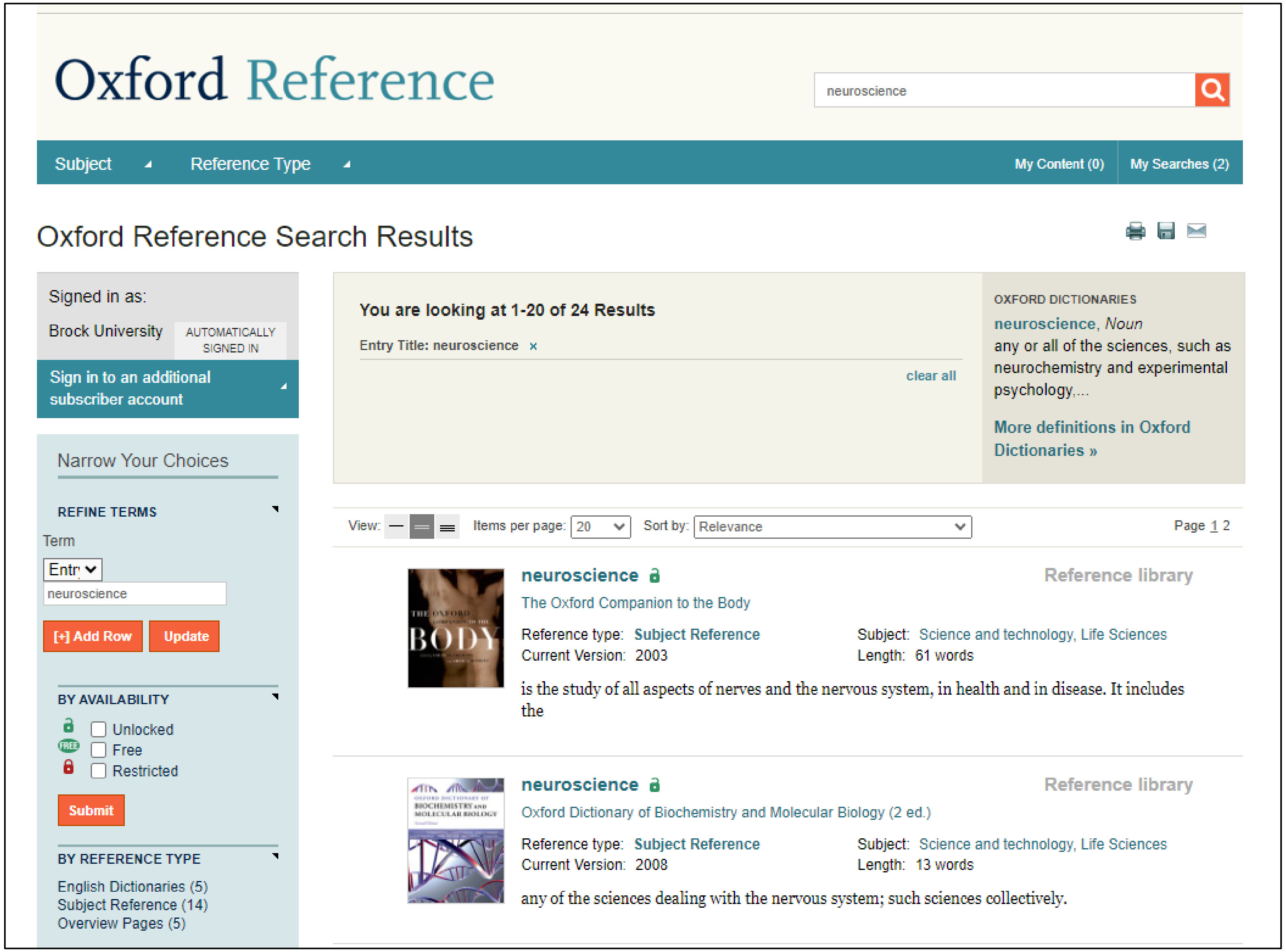

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OXFORD DICTIONARIES

neuroscience, *Noun*

any or all of the sciences, such as neurochemistry and experimental psychology,...

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neuroscience Reference library

The Oxford Companion to the Body

Reference type: Subject Reference Subject: Science and technology, Life Sciences

Current Version: 2003 Length: 61 words

is the study of all aspects of nerves and the nervous system, in health and in disease. It includes the

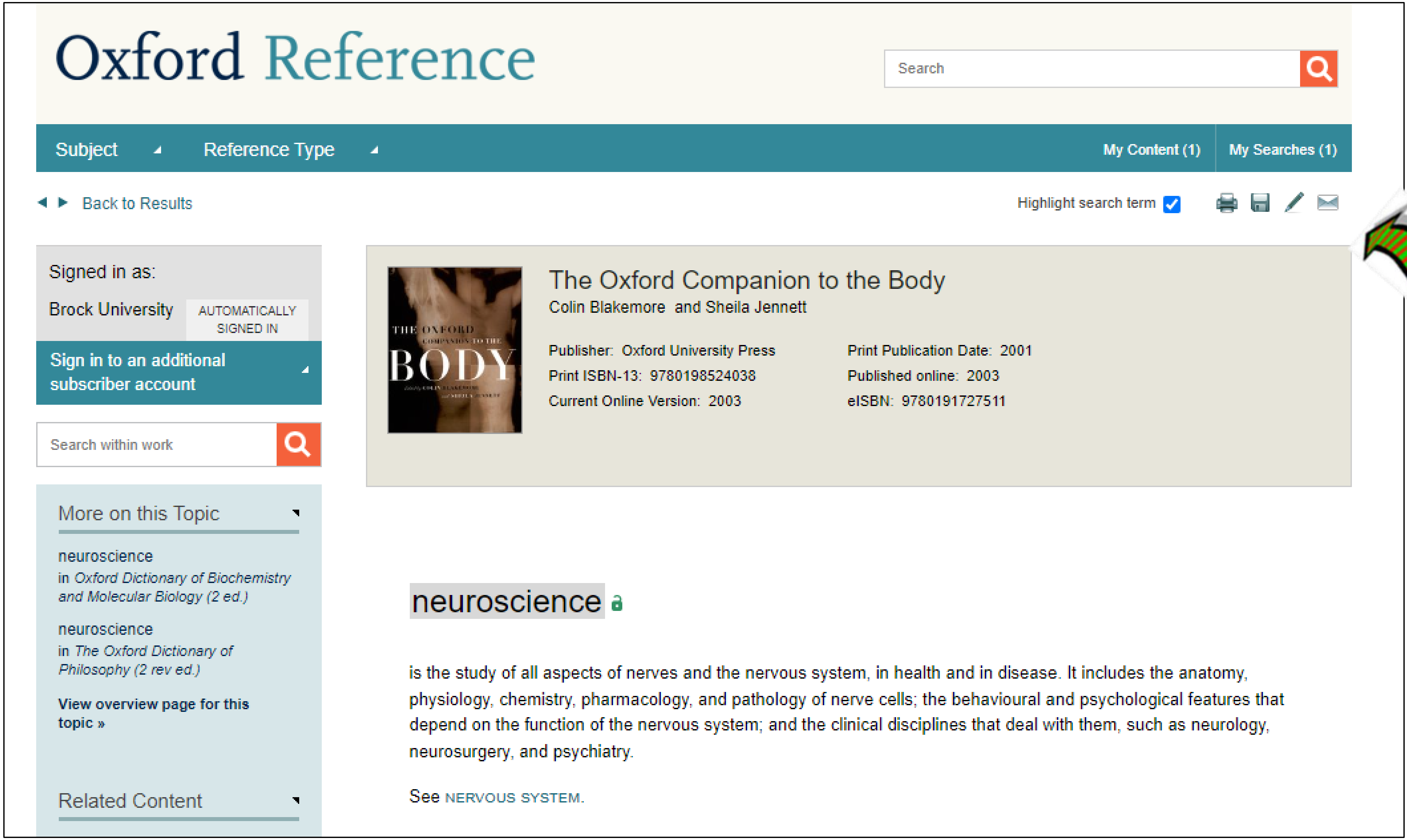
neuroscience Reference library

Oxford Dictionary of Biochemistry and Molecular Biology (2 ed.)

Reference type: Subject Reference Subject: Science and technology, Life Sciences

Current Version: 2008 Length: 13 words

any of the sciences dealing with the nervous system; such sciences collectively.



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More on this Topic

neuroscience
in *Oxford Dictionary of Biochemistry and Molecular Biology (2 ed.)*

neuroscience
in *The Oxford Dictionary of Philosophy (2 rev ed.)*

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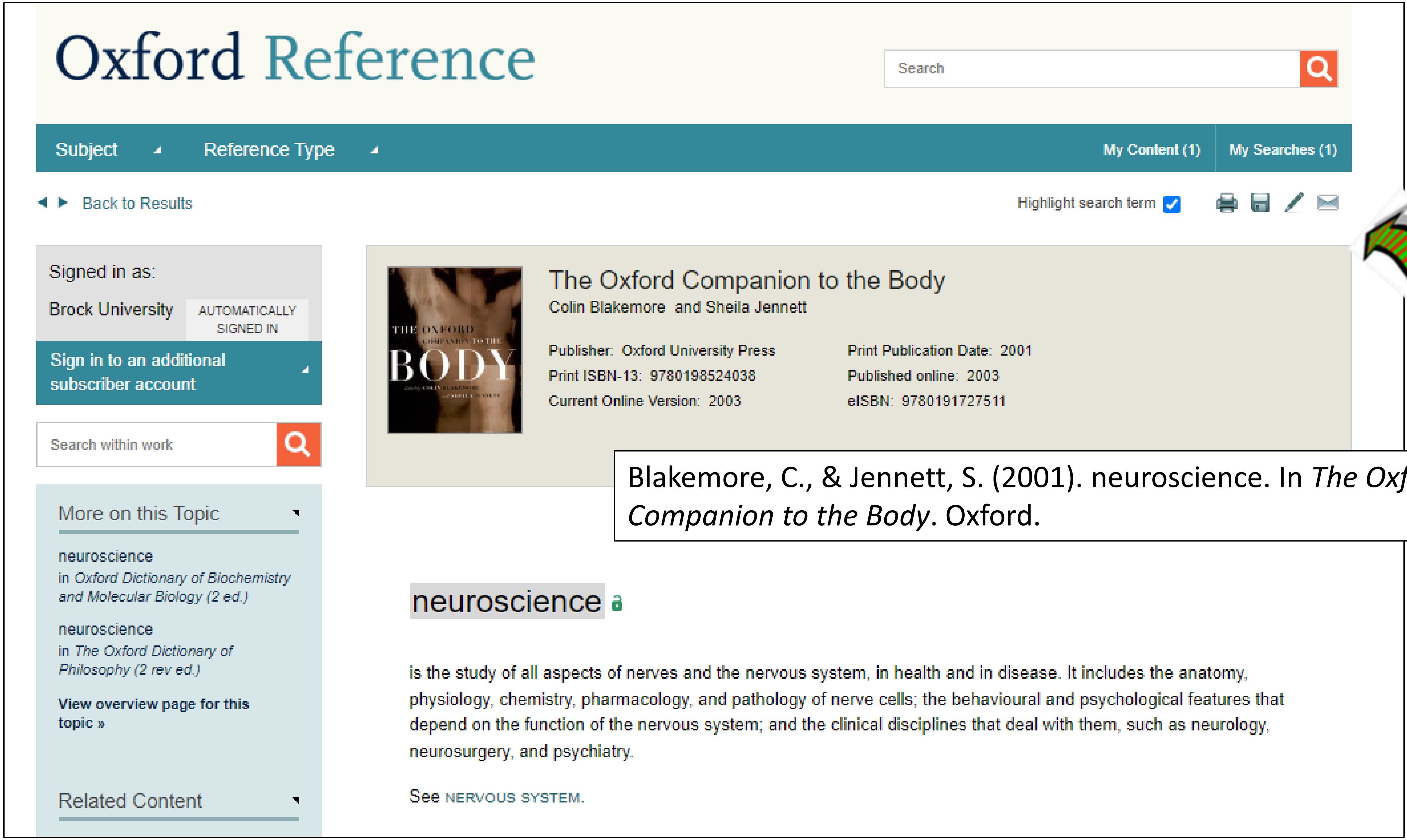
The Oxford Companion to the Body
Colin Blakemore and Sheila Jennett

Publisher: Oxford University Press	Print Publication Date: 2001
Print ISBN-13: 9780198524038	Published online: 2003
Current Online Version: 2003	eISBN: 9780191727511

neuroscience

is the study of all aspects of nerves and the nervous system, in health and in disease. It includes the anatomy, physiology, chemistry, pharmacology, and pathology of nerve cells; the behavioural and psychological features that depend on the function of the nervous system; and the clinical disciplines that deal with them, such as neurology, neurosurgery, and psychiatry.

See NERVOUS SYSTEM.



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Colin Blakemore and Sheila Jennett

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Print ISBN-13: 9780198524038
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neuroscience
in *Oxford Dictionary of Biochemistry and Molecular Biology (2 ed.)*

neuroscience
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Blakemore, C., & Jennett, S. (2001). neuroscience. In *The Oxford Companion to the Body*. Oxford.

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See NERVOUS SYSTEM.

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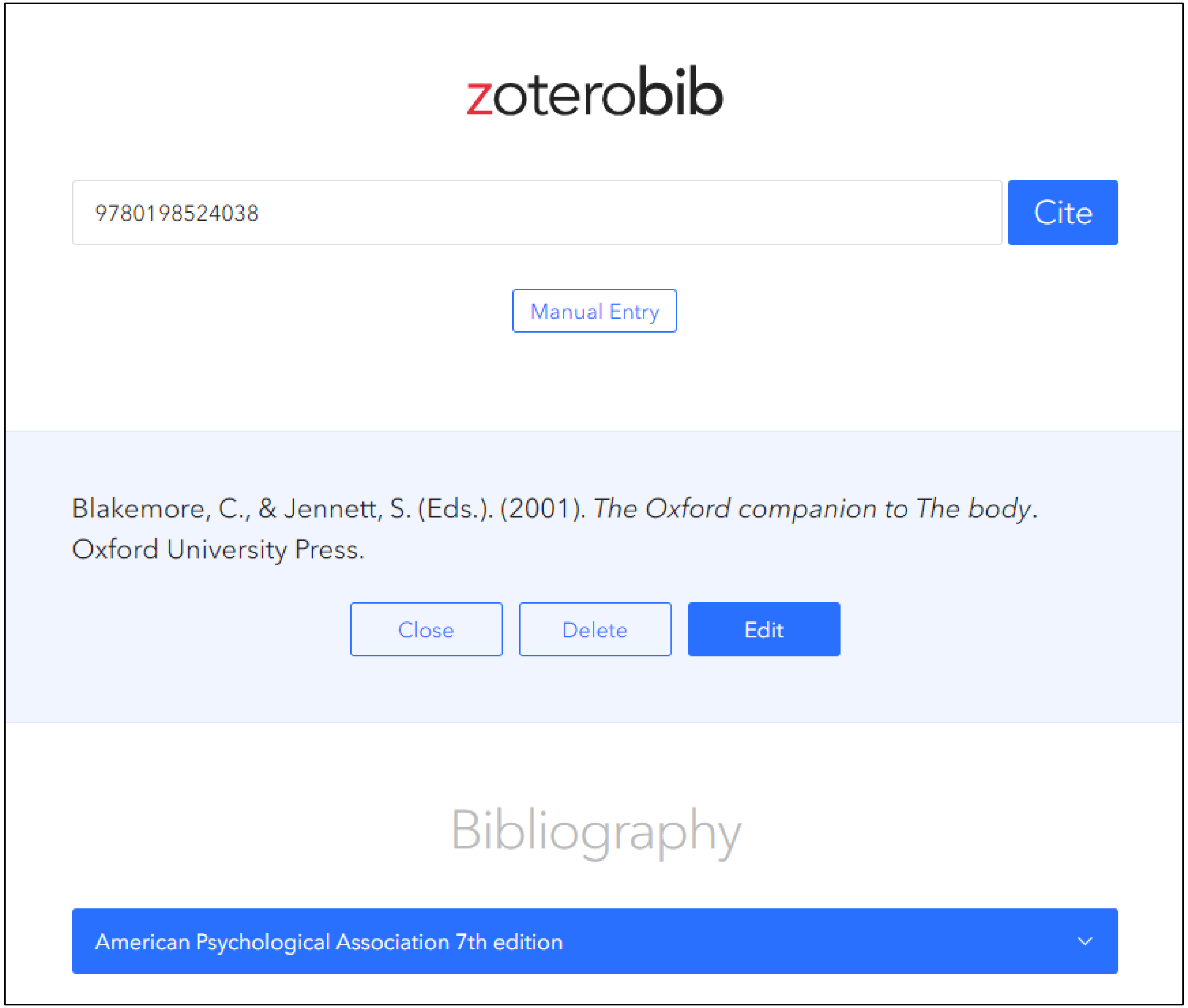

Blakemore, C., & Jennett, S. (Eds.). (2001). *The Oxford companion to The body*. Oxford University Press.

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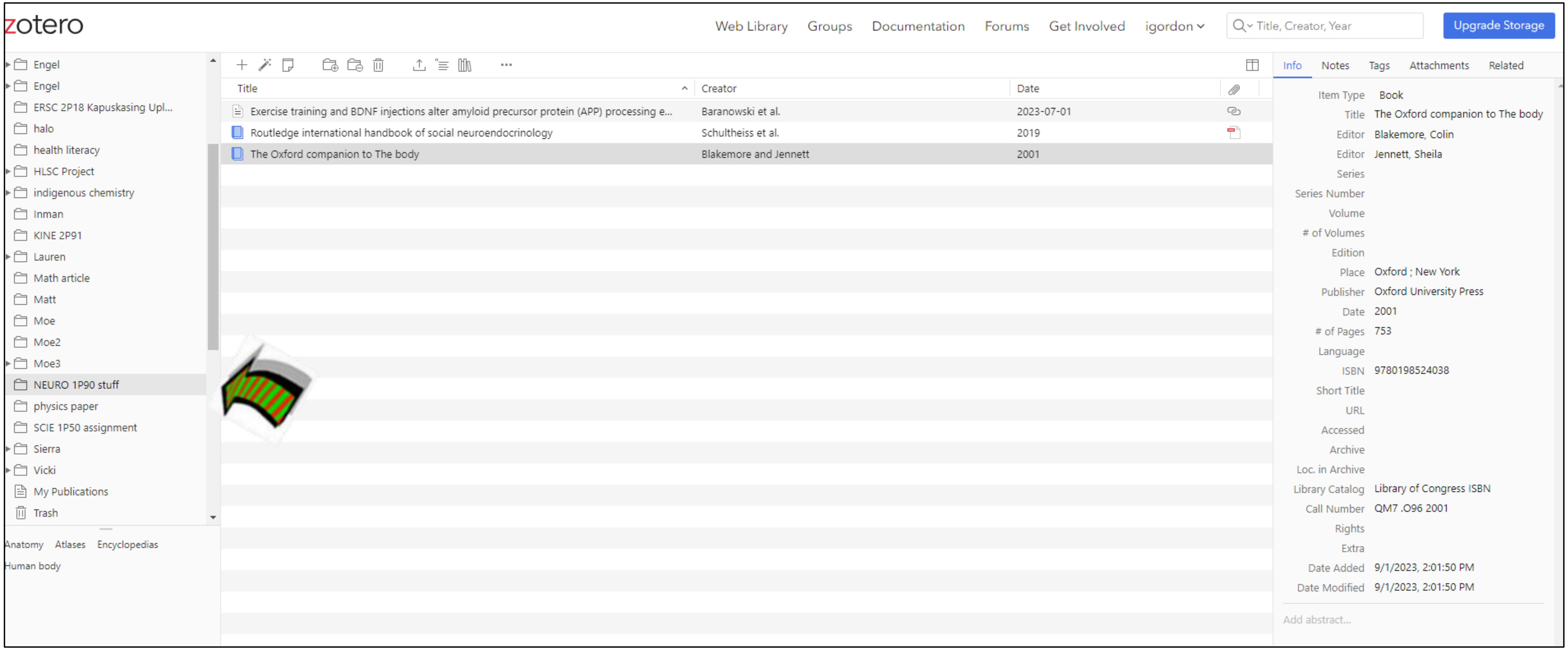

Title	Creator	Date
Exercise training and BDNF injections alter amyloid precursor protein (APP) processing e...	Baranowski et al.	2023-07-01
Routledge international handbook of social neuroendocrinology	Schultheiss et al.	2019
The Oxford companion to The body	Blakemore and Jennett	2001

Item Type	Book
Title	The Oxford companion to The body
Editor	Blakemore, Colin
Editor	Jennett, Sheila
Series	
Series Number	
Volume	
# of Volumes	
Edition	
Place	Oxford ; New York
Publisher	Oxford University Press
Date	2001
# of Pages	753
Language	
ISBN	9780198524038
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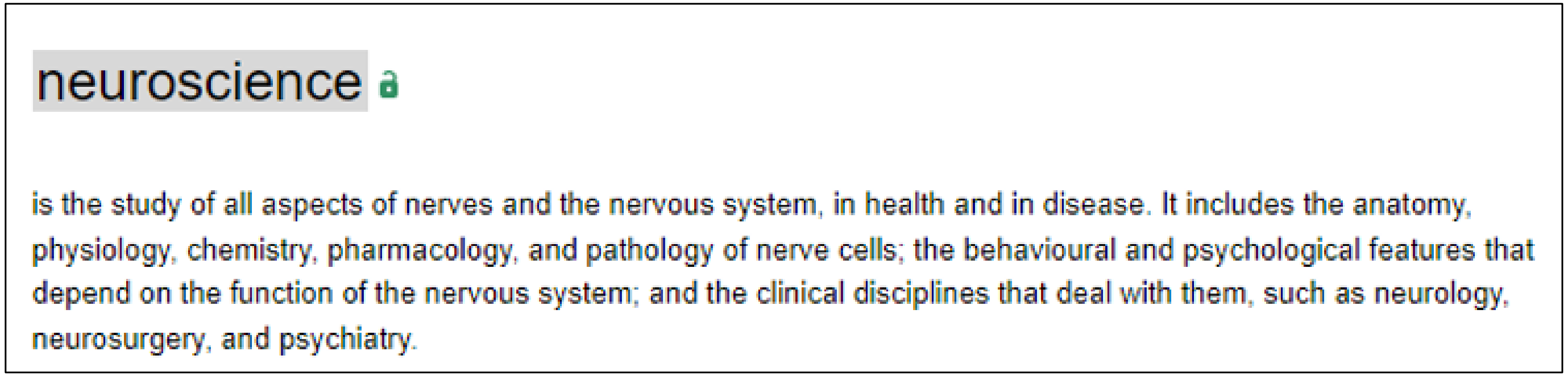

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Blakemore & Jennett (2001) define neuroscience as “…the study of all aspects of nerves and the nervous system…”

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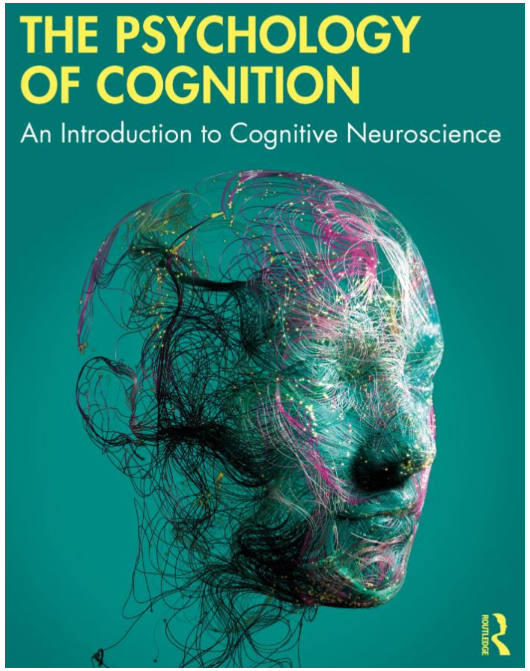

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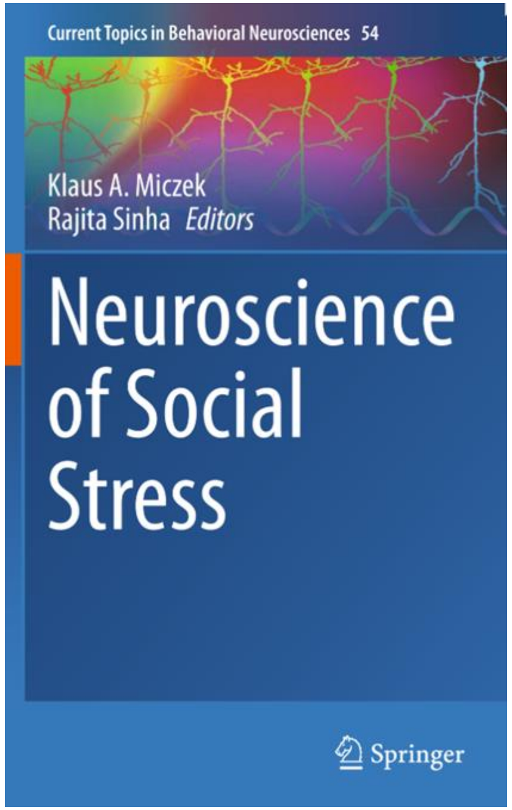

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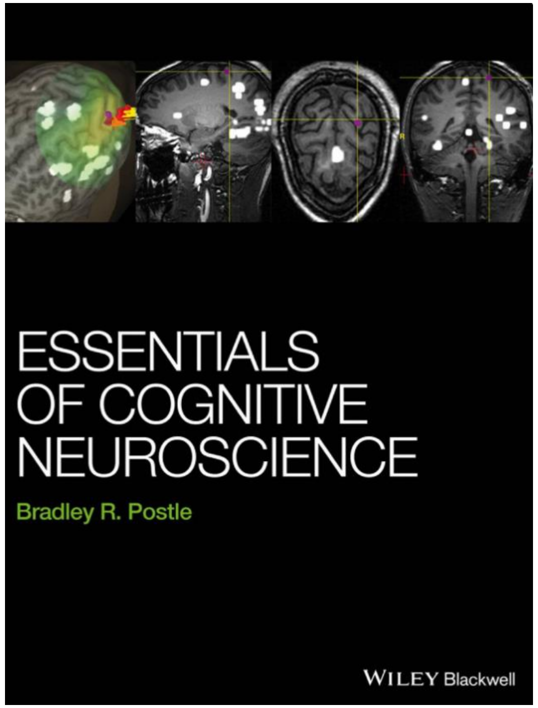

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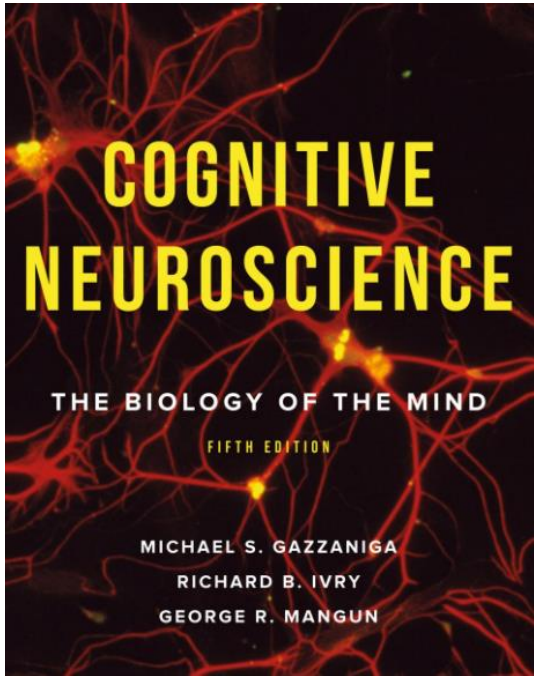

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THE BIOLOGY OF THE MIND
FIFTH EDITION

MICHAEL S. GAZZANIGA
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Miczek, K. A., & Sinha, R. (Eds.). (2022). *Neuroscience of social stress*. Springer.

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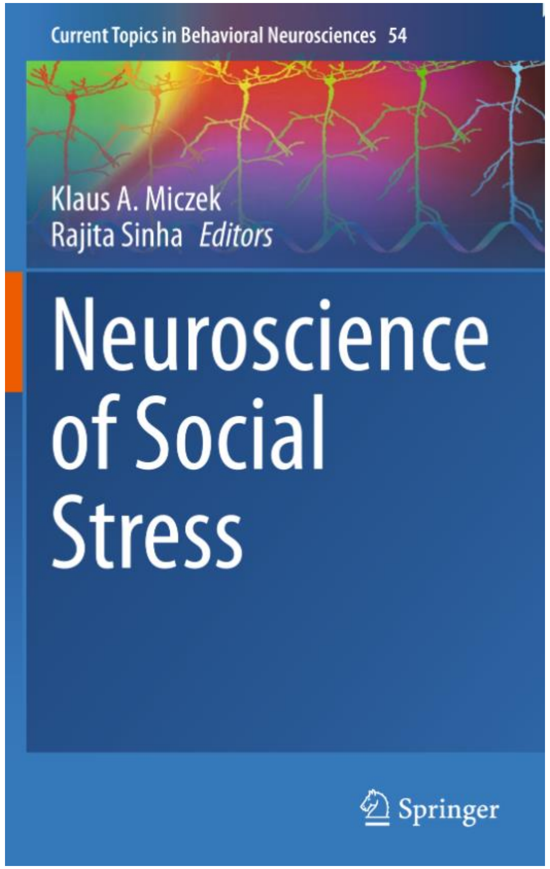
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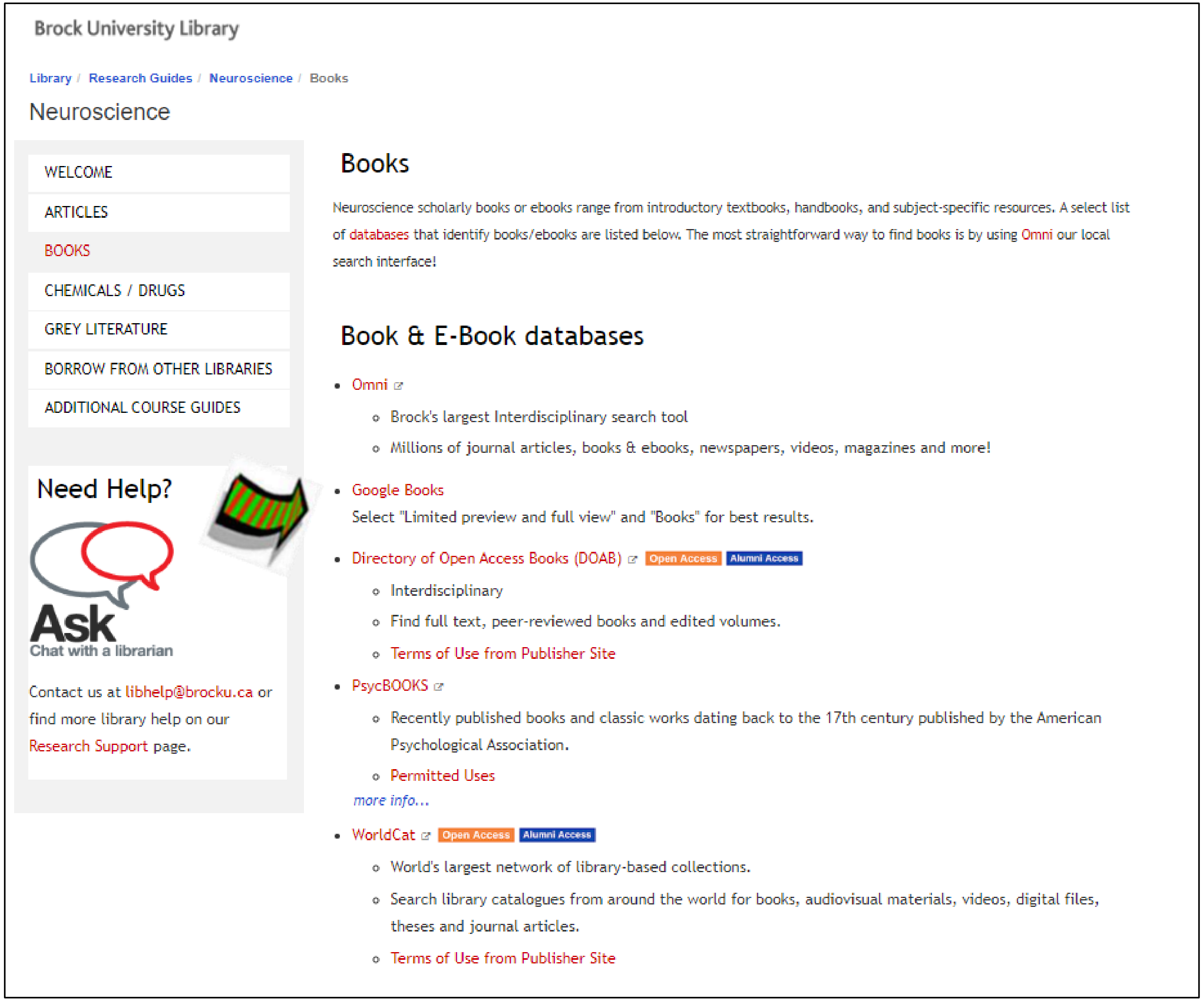

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Books

Neuroscience scholarly books or ebooks range from introductory textbooks, handbooks, and subject-specific resources. A select list of databases that identify books/ebooks are listed below. The most straightforward way to find books is by using Omni our local search interface!

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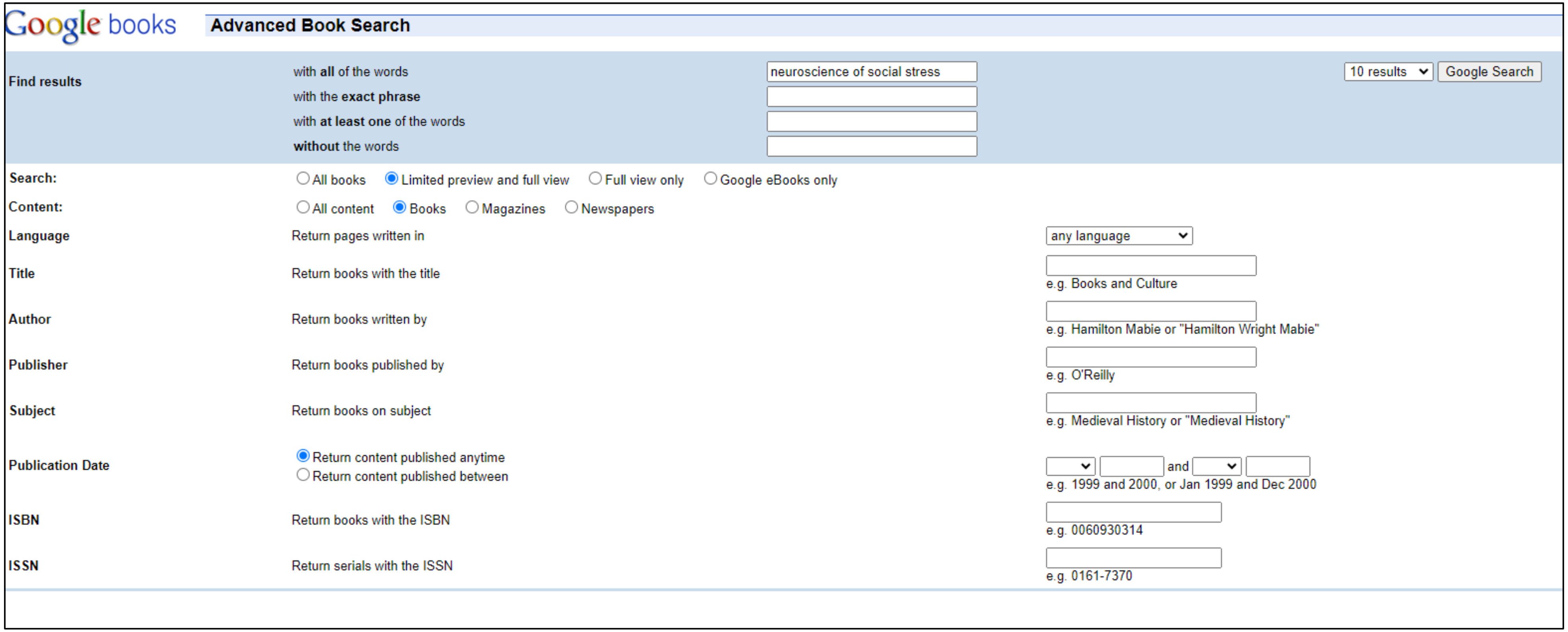

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Methods and Challenges in Investigating Sex-Specific Consequences of Social Stressors in Adolescence in Rats: Is It the Stress or the Social or the Stage of Development?

Cheryl M. McCormick

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Abstract Adolescence is a time of social learning and social restructuring that is accompanied by changes in both the hypothalamic-pituitary-gonadal axis and the hypothalamic-pituitary-adrenal (HPA) axis. The activation of these axes by puberty

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e-mail: cmccormick@brocku.ca

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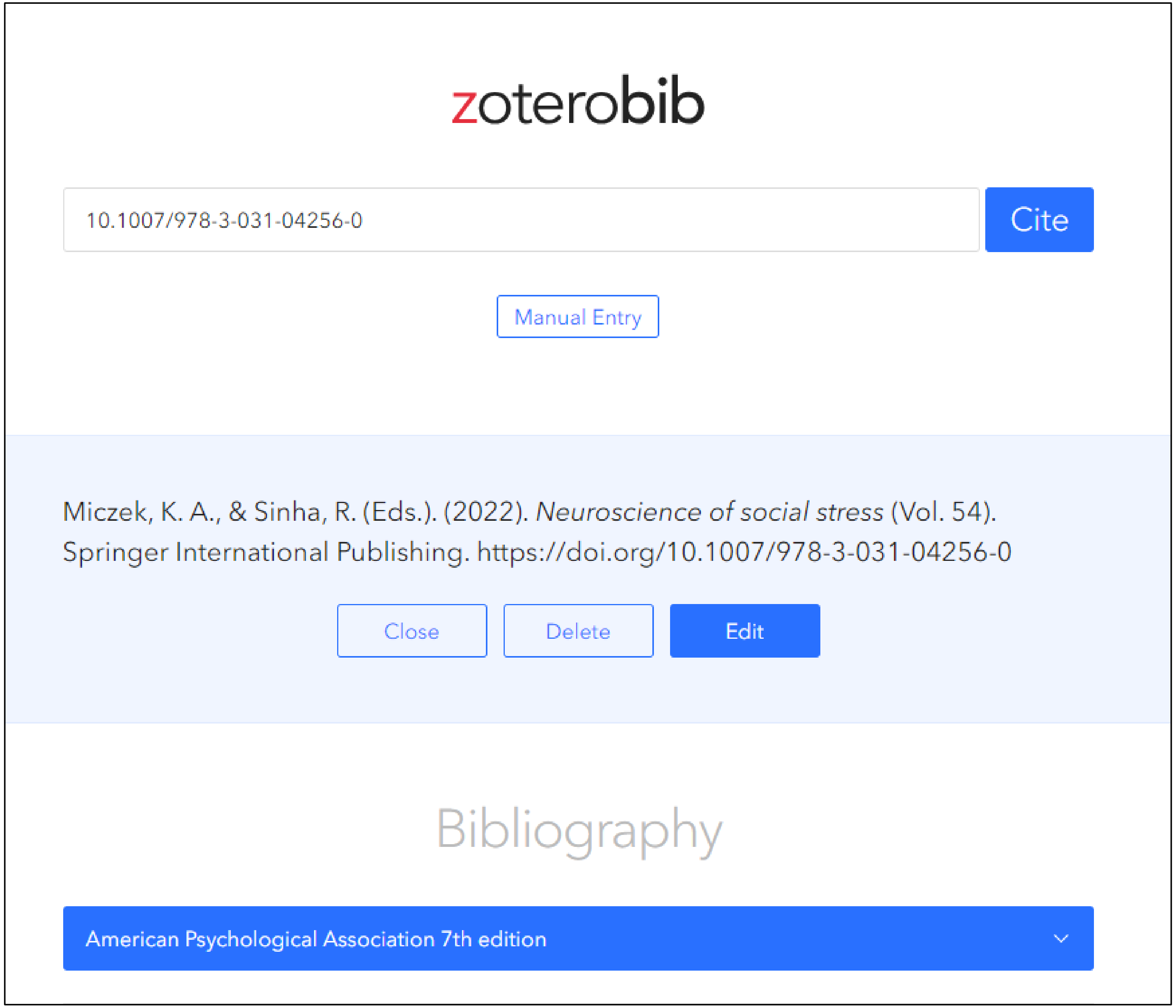

10.1007/978-3-031-04256-0

Miczek, K. A., & Sinha, R. (Eds.). (2022). *Neuroscience of social stress* (Vol. 54). Springer International Publishing. https://doi.org/10.1007/978-3-031-04256-0

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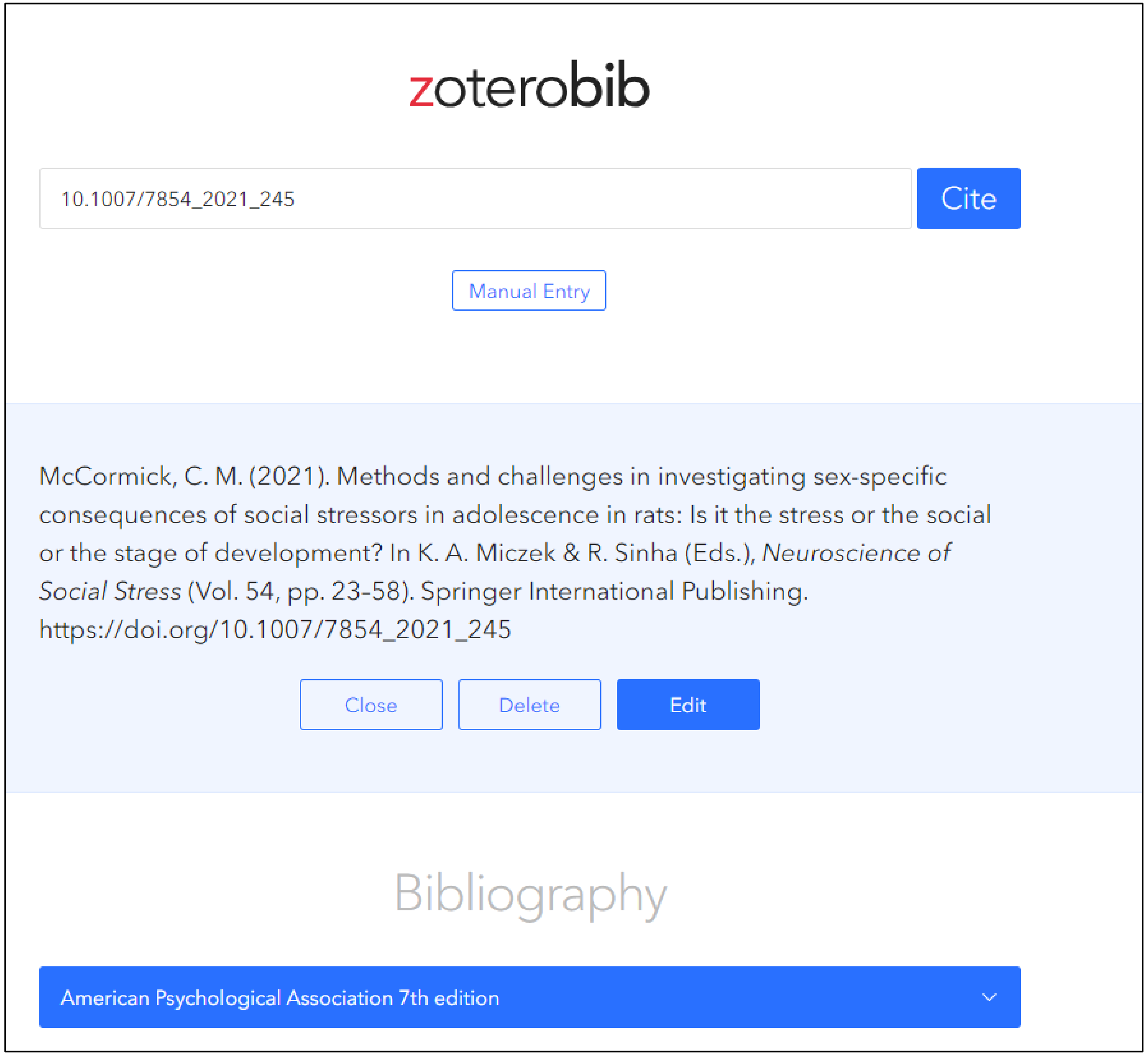

10.1007/7854_2021_245

McCormick, C. M. (2021). Methods and challenges in investigating sex-specific consequences of social stressors in adolescence in rats: Is it the stress or the social or the stage of development? In K. A. Miczek & R. Sinha (Eds.), *Neuroscience of Social Stress* (Vol. 54, pp. 23-58). Springer International Publishing. https://doi.org/10.1007/7854_2021_245

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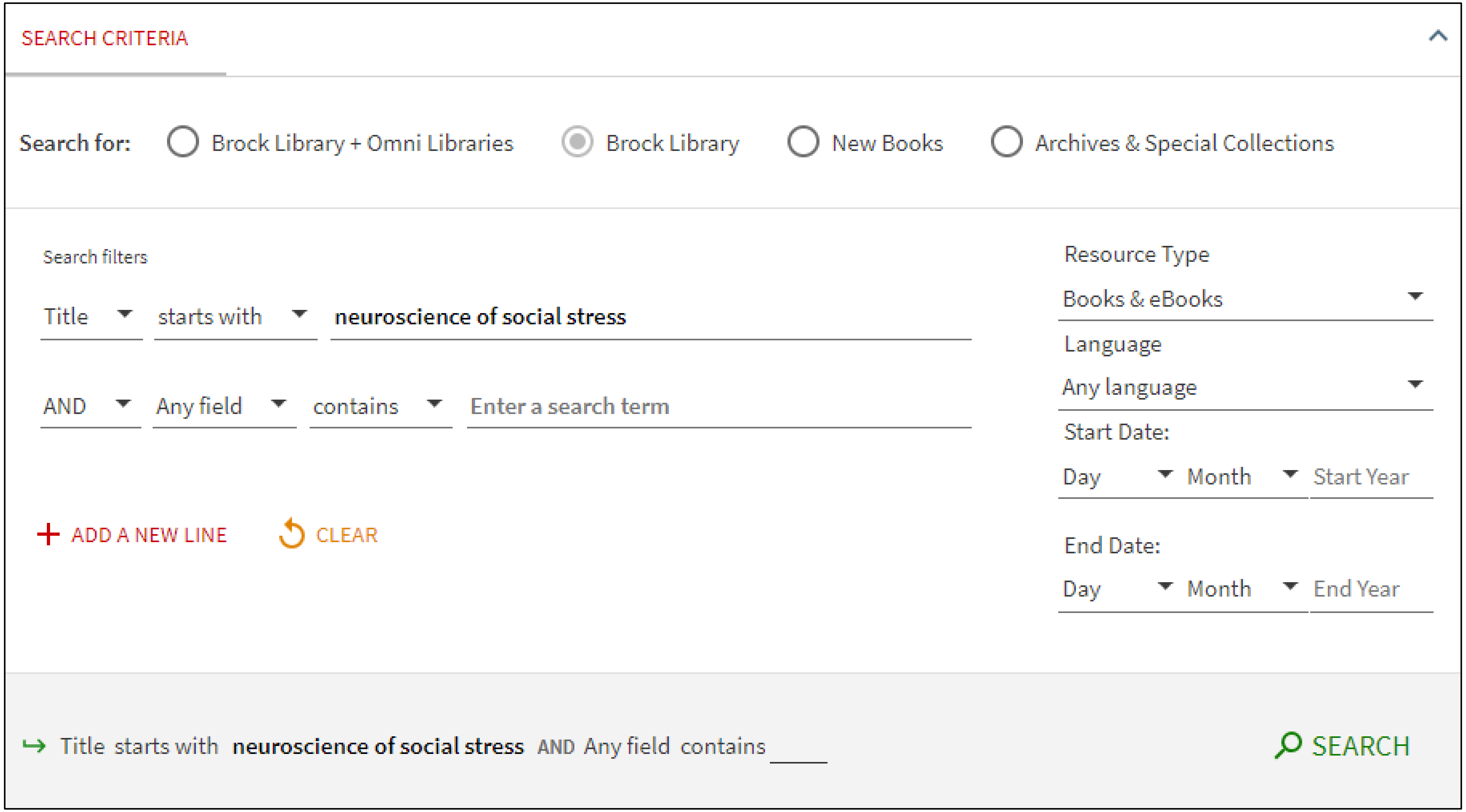

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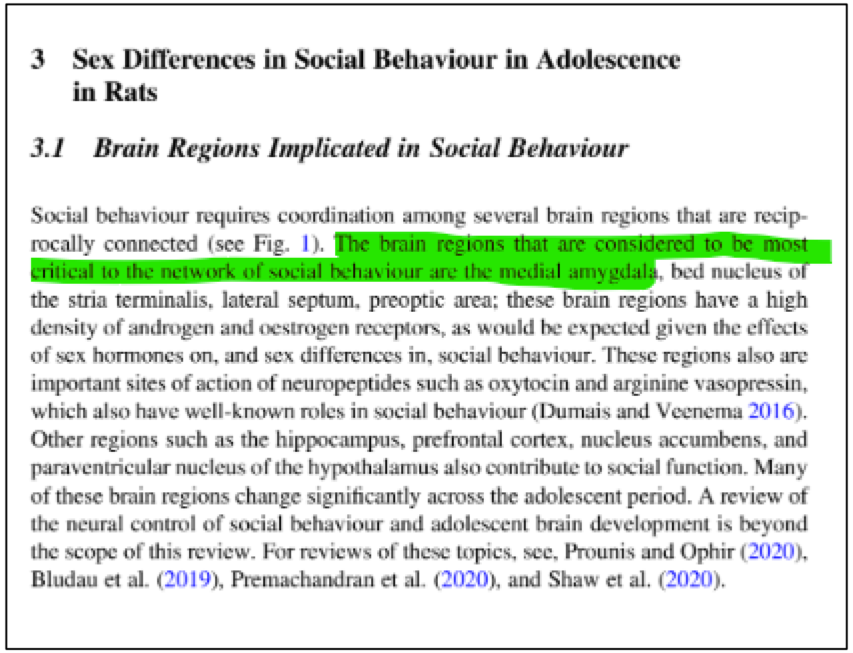

3 Sex Differences in Social Behaviour in Adolescence in Rats

3.1 *Brain Regions Implicated in Social Behaviour*

Social behaviour requires coordination among several brain regions that are reciprocally connected (see Fig. 1). The brain regions that are considered to be most critical to the network of social behaviour are the medial amygdala, bed nucleus of the stria terminalis, lateral septum, preoptic area; these brain regions have a high density of androgen and oestrogen receptors, as would be expected given the effects of sex hormones on, and sex differences in, social behaviour. These regions also are important sites of action of neuropeptides such as oxytocin and arginine vasopressin, which also have well-known roles in social behaviour (Dumais and Veenema 2016). Other regions such as the hippocampus, prefrontal cortex, nucleus accumbens, and paraventricular nucleus of the hypothalamus also contribute to social function. Many of these brain regions change significantly across the adolescent period. A review of the neural control of social behaviour and adolescent brain development is beyond the scope of this review. For reviews of these topics, see, Prounis and Ophir (2020), Bludau et al. (2019), Premachandran et al. (2020), and Shaw et al. (2020).

In this lab I observed that "….The brain regions that are considered to be most critical to the network of social behaviour are the medial amygdala…" (McCormick, 2001, 27).

McCormick, C. M. (2021). Methods and challenges in investigating sex-specific consequences of social stressors in adolescence in rats: Is it the stress or the social or the stage of development? In K. A. Miczek & R. Sinha (Eds.), *Neuroscience of Social Stress* (Vol. 54, pp. 23–58). Springer. https://doi.org/10.1007/7854_2021_245

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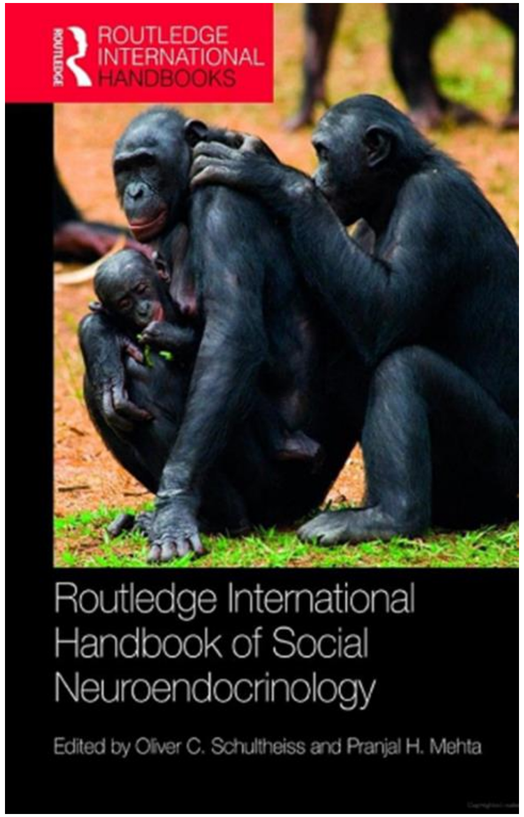
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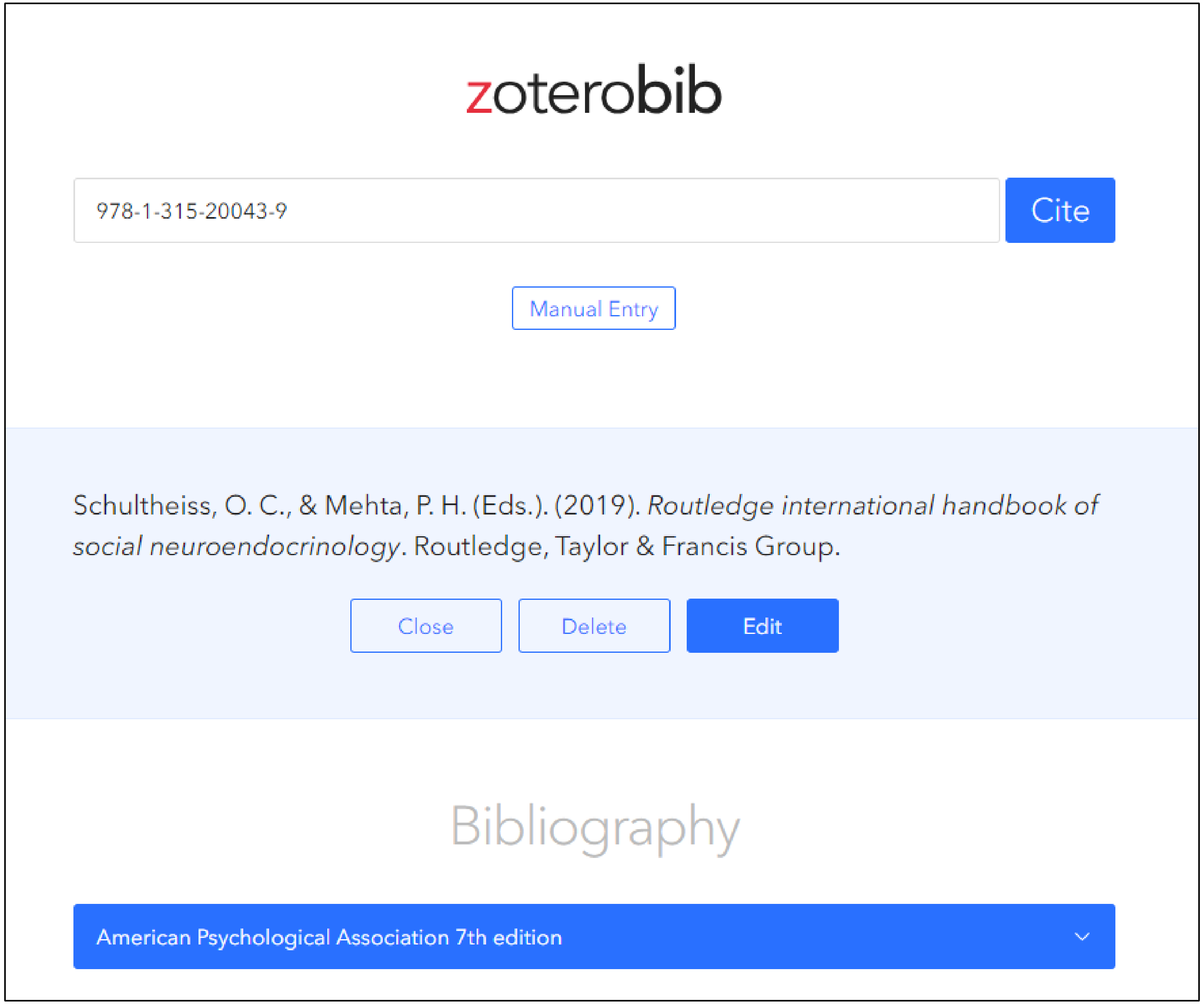

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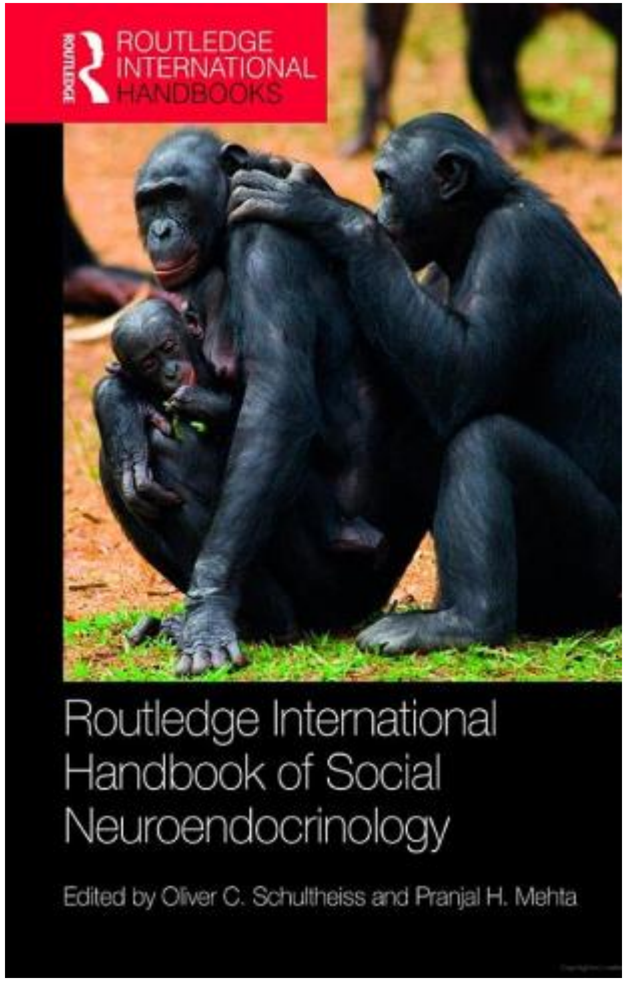
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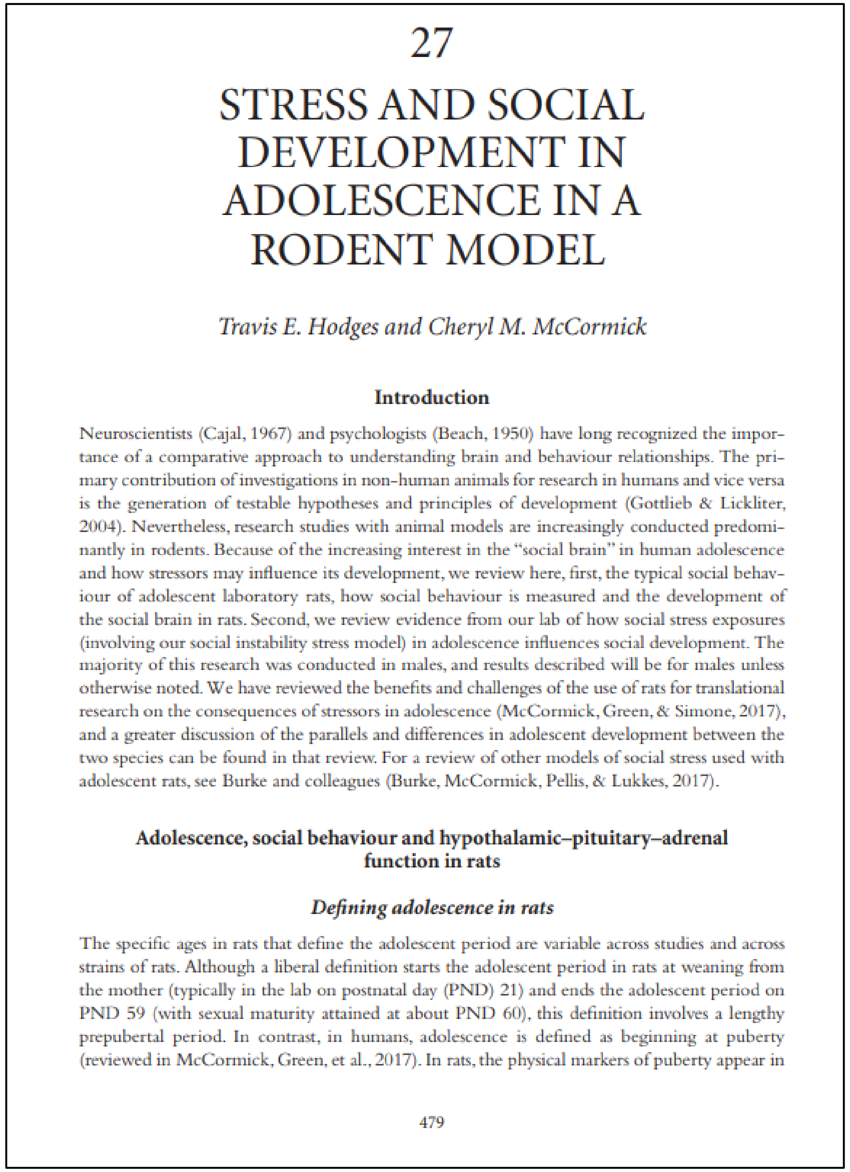


27

STRESS AND SOCIAL DEVELOPMENT IN ADOLESCENCE IN A RODENT MODEL

Travis E. Hodges and Cheryl M. McCormick

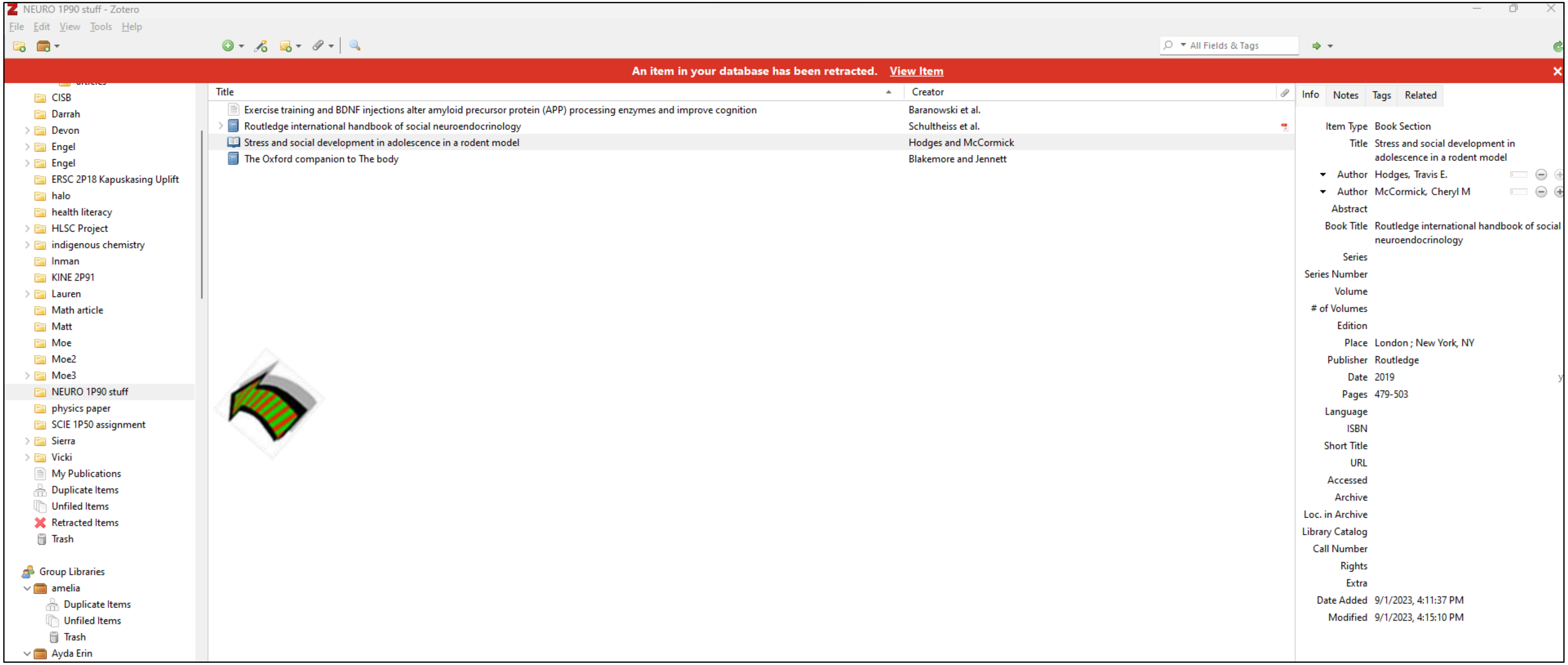
Introduction

Neuroscientists (Cajal, 1967) and psychologists (Beach, 1950) have long recognized the importance of a comparative approach to understanding brain and behaviour relationships. The primary contribution of investigations in non-human animals for research in humans and vice versa is the generation of testable hypotheses and principles of development (Gottlieb & Lickliter, 2004). Nevertheless, research studies with animal models are increasingly conducted predominantly in rodents. Because of the increasing interest in the "social brain" in human adolescence and how stressors may influence its development, we review here, first, the typical social behaviour of adolescent laboratory rats, how social behaviour is measured and the development of the social brain in rats. Second, we review evidence from our lab of how social stress exposures (involving our social instability stress model) in adolescence influences social development. The majority of this research was conducted in males, and results described will be for males unless otherwise noted. We have reviewed the benefits and challenges of the use of rats for translational research on the consequences of stressors in adolescence (McCormick, Green, & Simone, 2017), and a greater discussion of the parallels and differences in adolescent development between the two species can be found in that review. For a review of other models of social stress used with adolescent rats, see Burke and colleagues (Burke, McCormick, Pellis, & Lukkes, 2017).

Adolescence, social behaviour and hypothalamic–pituitary–adrenal function in rats

Defining adolescence in rats

The specific ages in rats that define the adolescent period are variable across studies and across strains of rats. Although a liberal definition starts the adolescent period in rats at weaning from the mother (typically in the lab on postnatal day (PND) 21) and ends the adolescent period on PND 59 (with sexual maturity attained at about PND 60), this definition involves a lengthy prepubertal period. In contrast, in humans, adolescence is defined as beginning at puberty (reviewed in McCormick, Green, et al., 2017). In rats, the physical markers of puberty appear in



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The Oxford companion to The body	Blakemore and Jennett

Item Type	Book Section
Title	Stress and social development in adolescence in a rodent model
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Book Title	Routledge international handbook of social neuroendocrinology
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Series Number	
Volume	
# of Volumes	
Edition	
Place	London ; New York, NY
Publisher	Routledge
Date	2019
Pages	479-503
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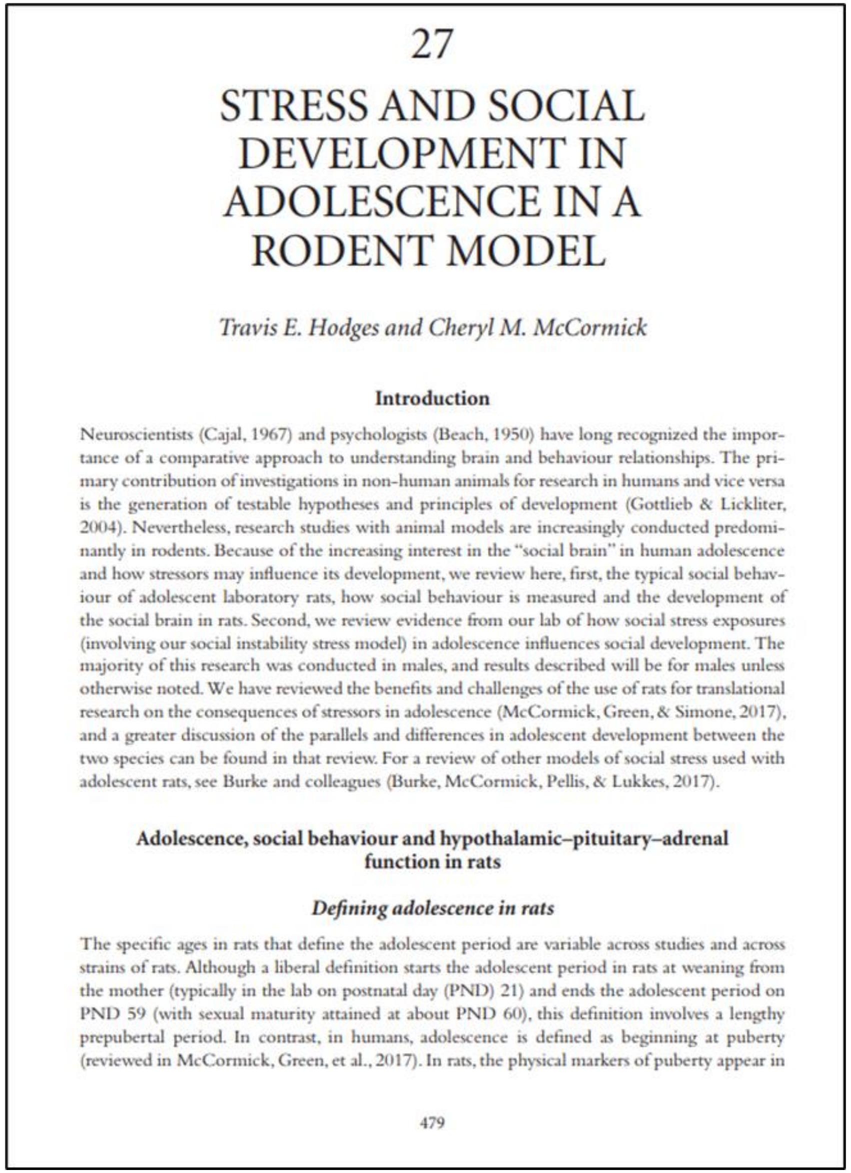

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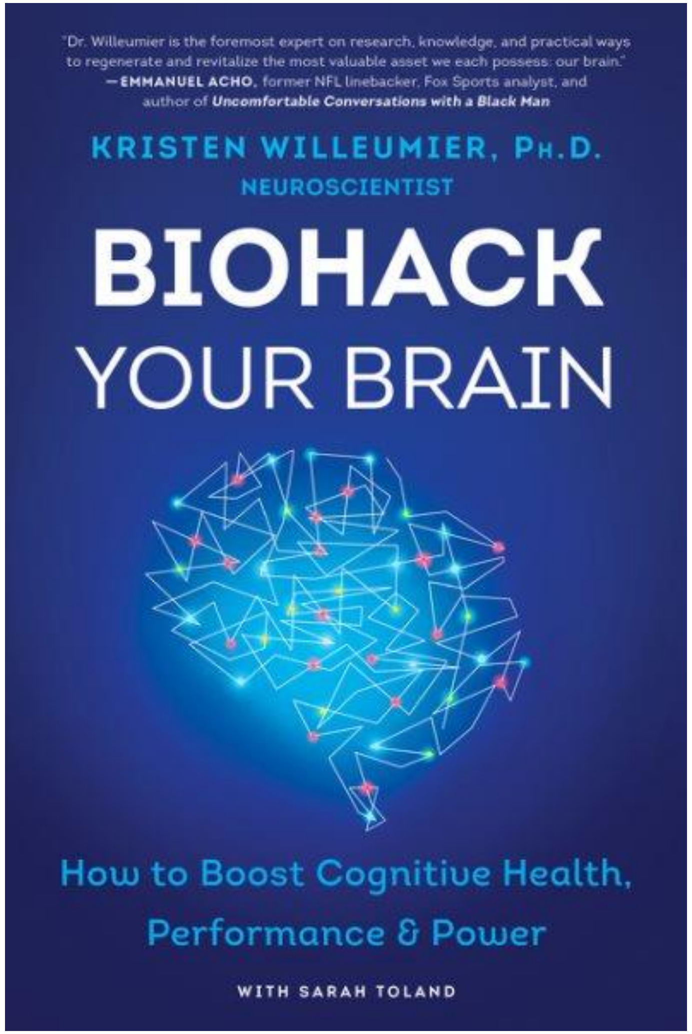
Willeumier, K., & Toland, S. (2020). *Biohack your brain: How to boost cognitive health, performance & power*. HarperCollins.


"Dr. Willeumier is the foremost expert on research, knowledge, and practical ways to regenerate and revitalize the most valuable asset we each possess: our brain."
—EMMANUEL ACHO, former NFL linebacker, Fox Sports analyst, and author of Uncomfortable Conversations with a Black Man
KRISTEN WILLEUMIER, PH.D.
NEUROSCIENTIST
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How to Boost Cognitive Health, Performance & Power
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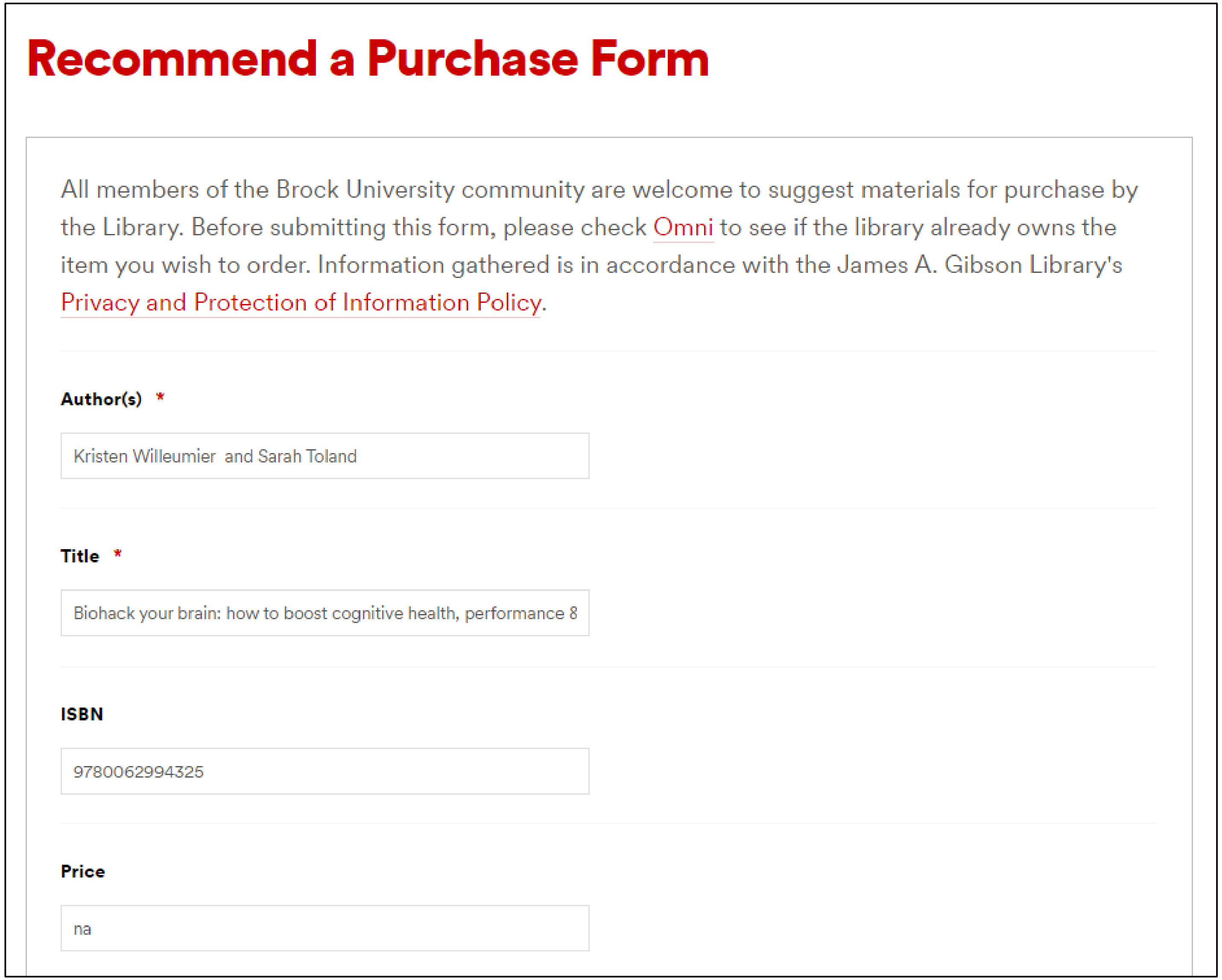

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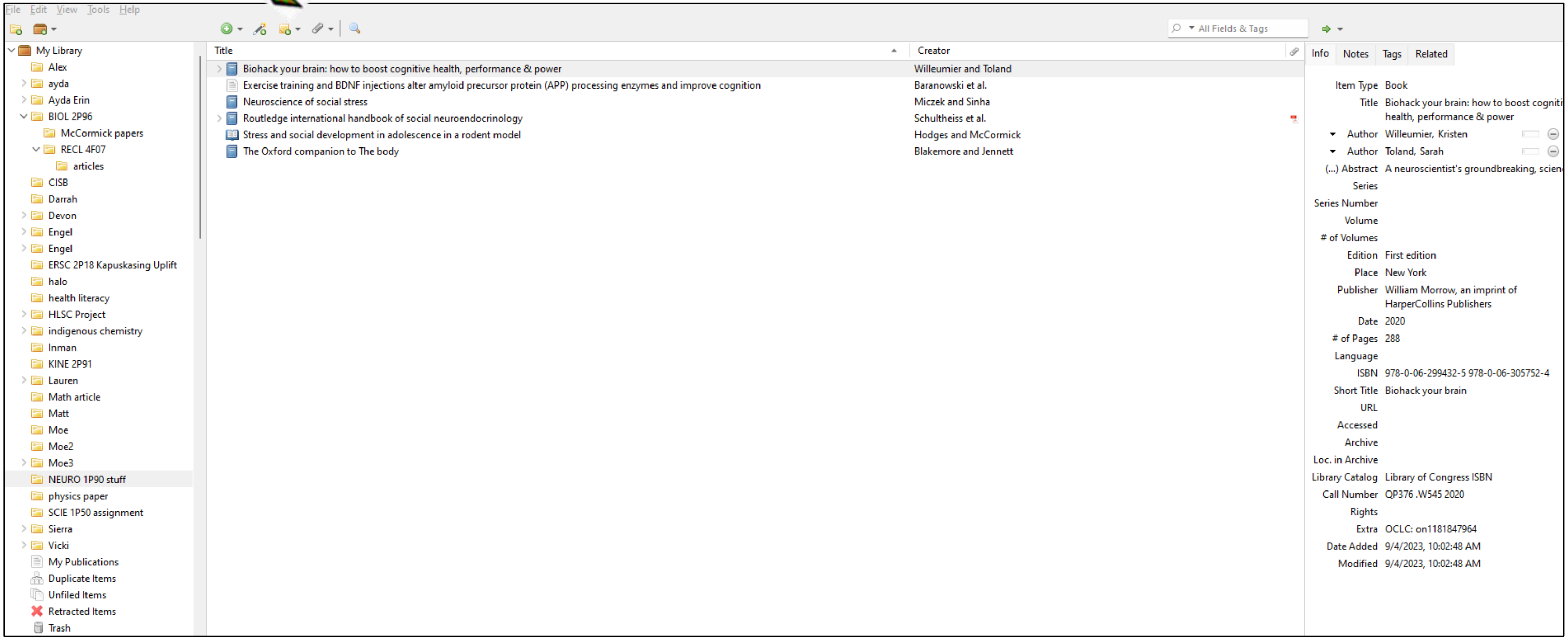

Title	Creator
Biohack your brain: how to boost cognitive health, performance & power	Willeumier and Toland
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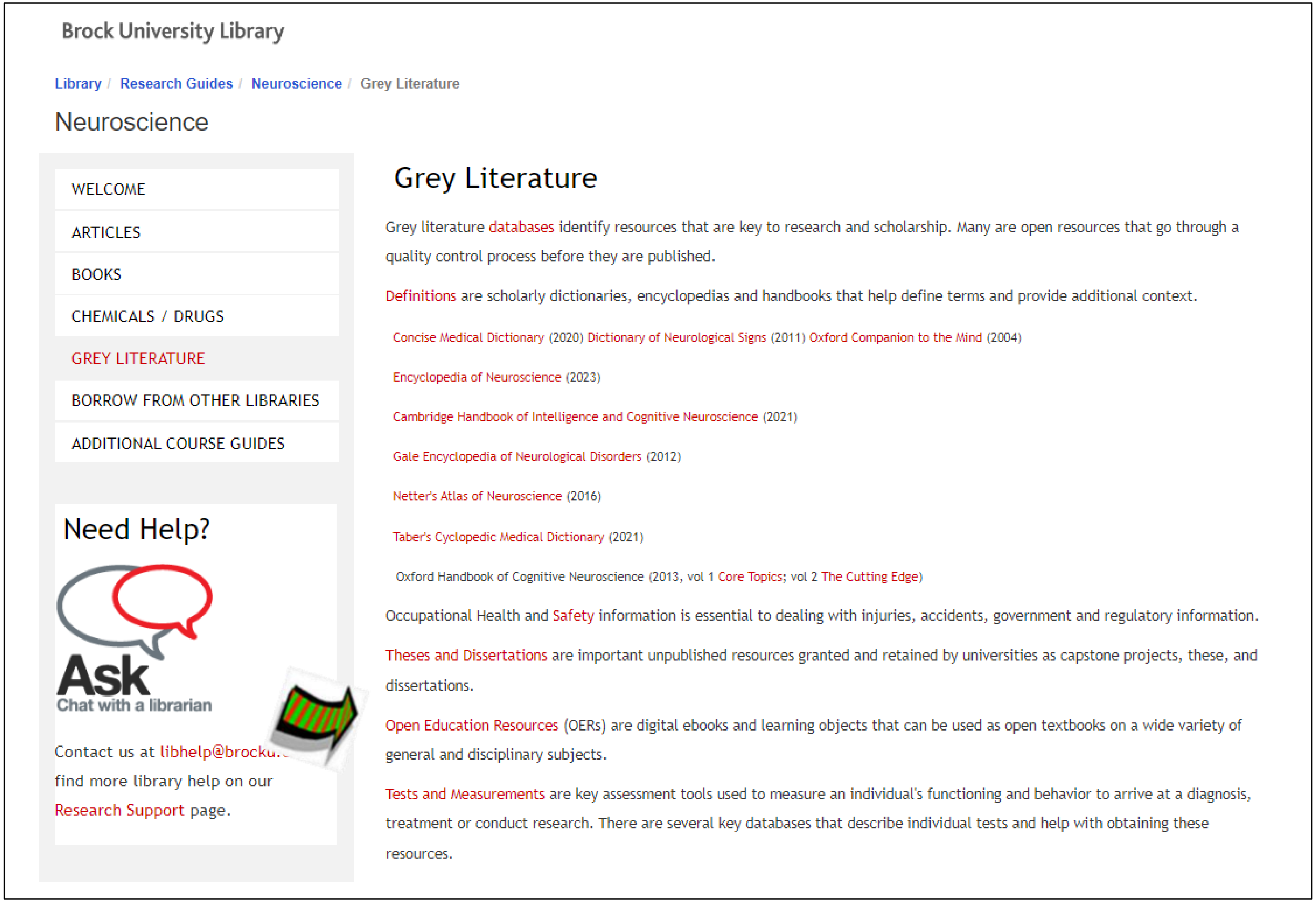

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Netter's Atlas of Neuroscience (2016)

Taber's Cyclopedic Medical Dictionary (2021)

Oxford Handbook of Cognitive Neuroscience (2013, vol 1 Core Topics; vol 2 The Cutting Edge)

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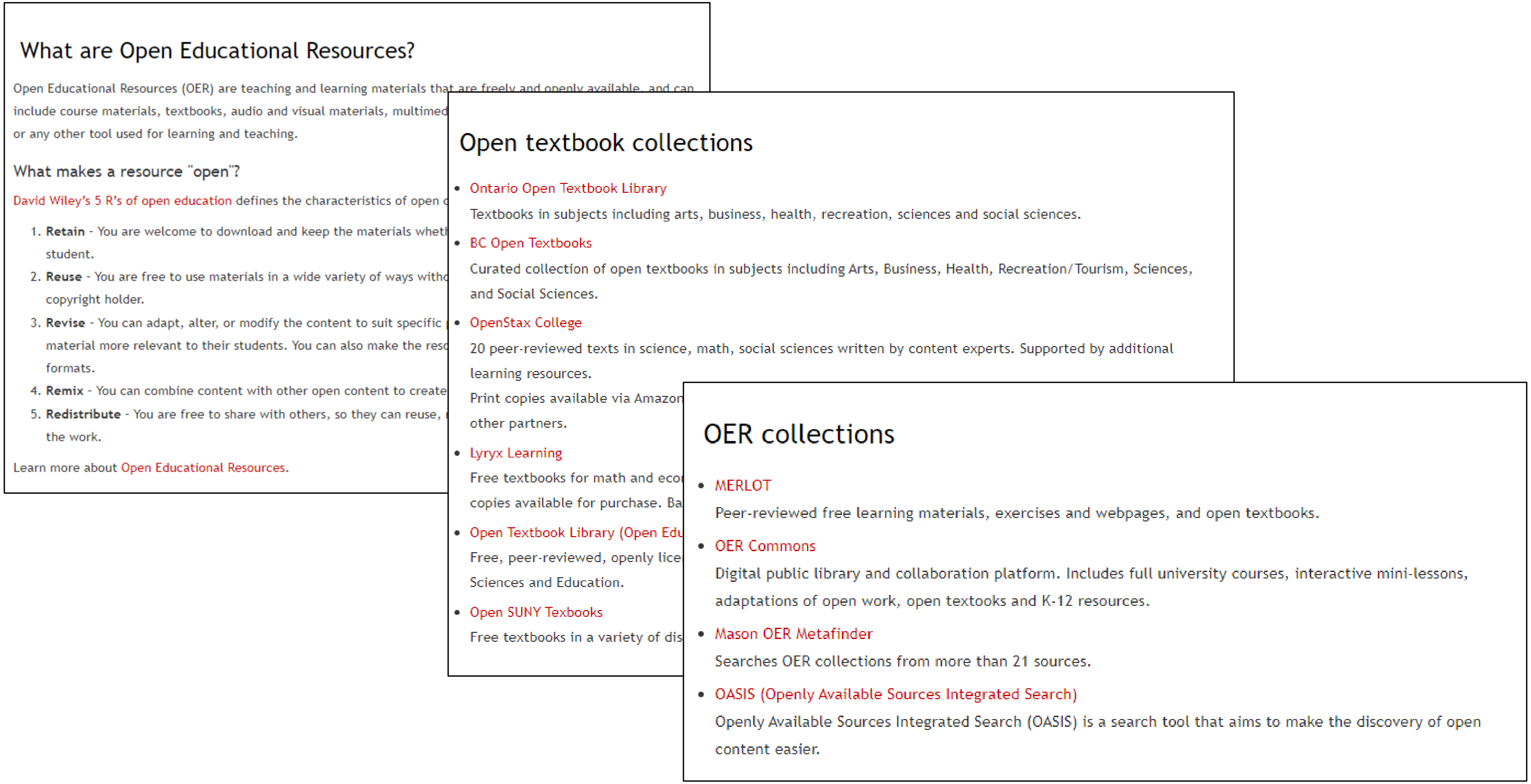

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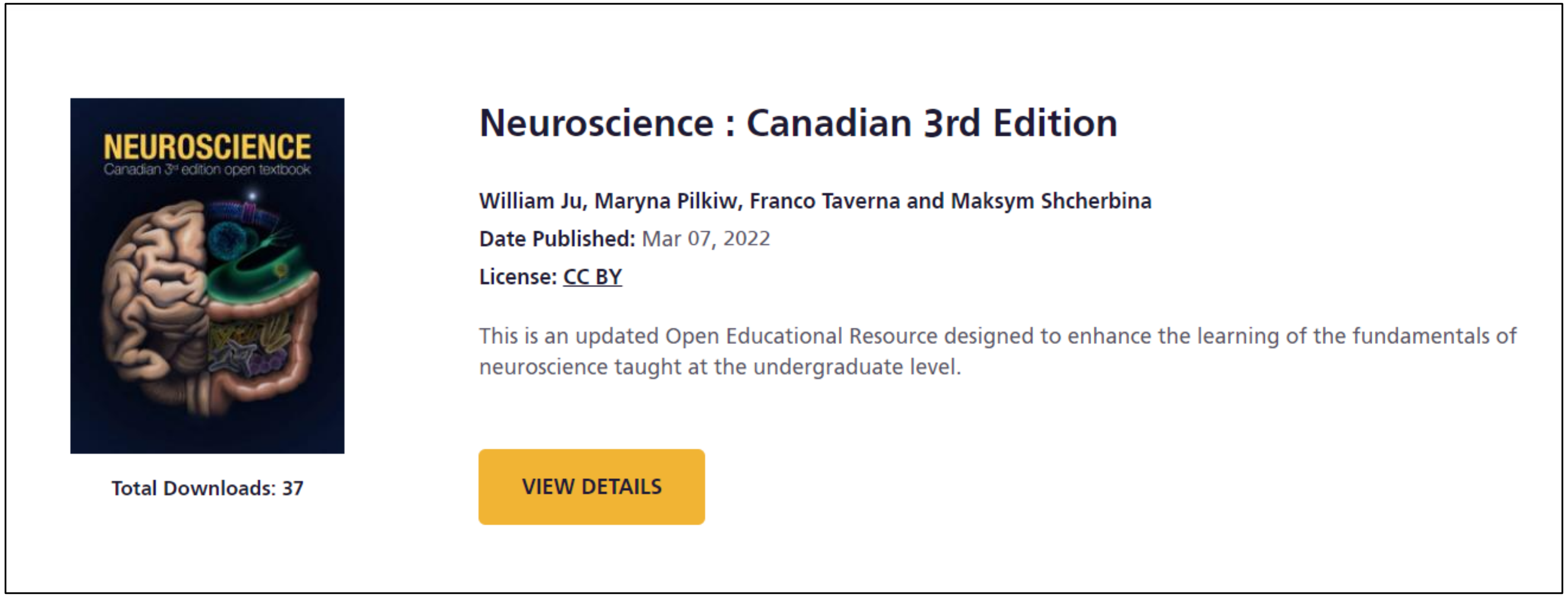

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William Ju, Maryna Pilkiw, Franco Taverna and Maksym Shcherbina

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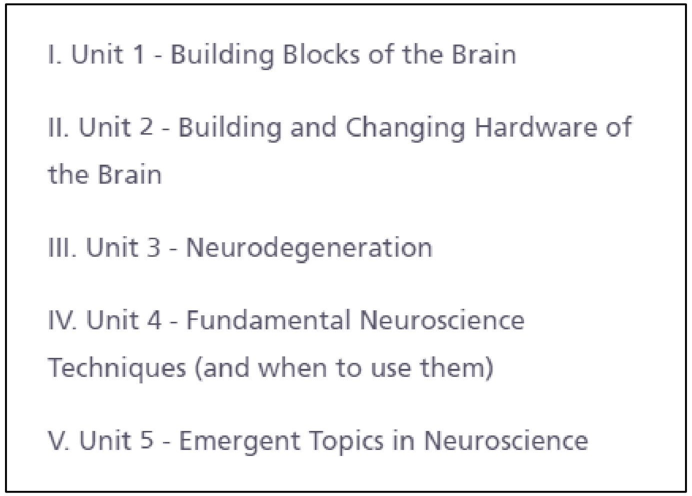
This is an updated Open Educational Resource designed to enhance the learning of the fundamentals of neuroscience taught at the undergraduate level.



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II. Unit 2 - Building and Changing Hardware of the Brain

III. Unit 3 - Neurodegeneration

IV. Unit 4 - Fundamental Neuroscience Techniques (and when to use them)

V. Unit 5 - Emergent Topics in Neuroscience

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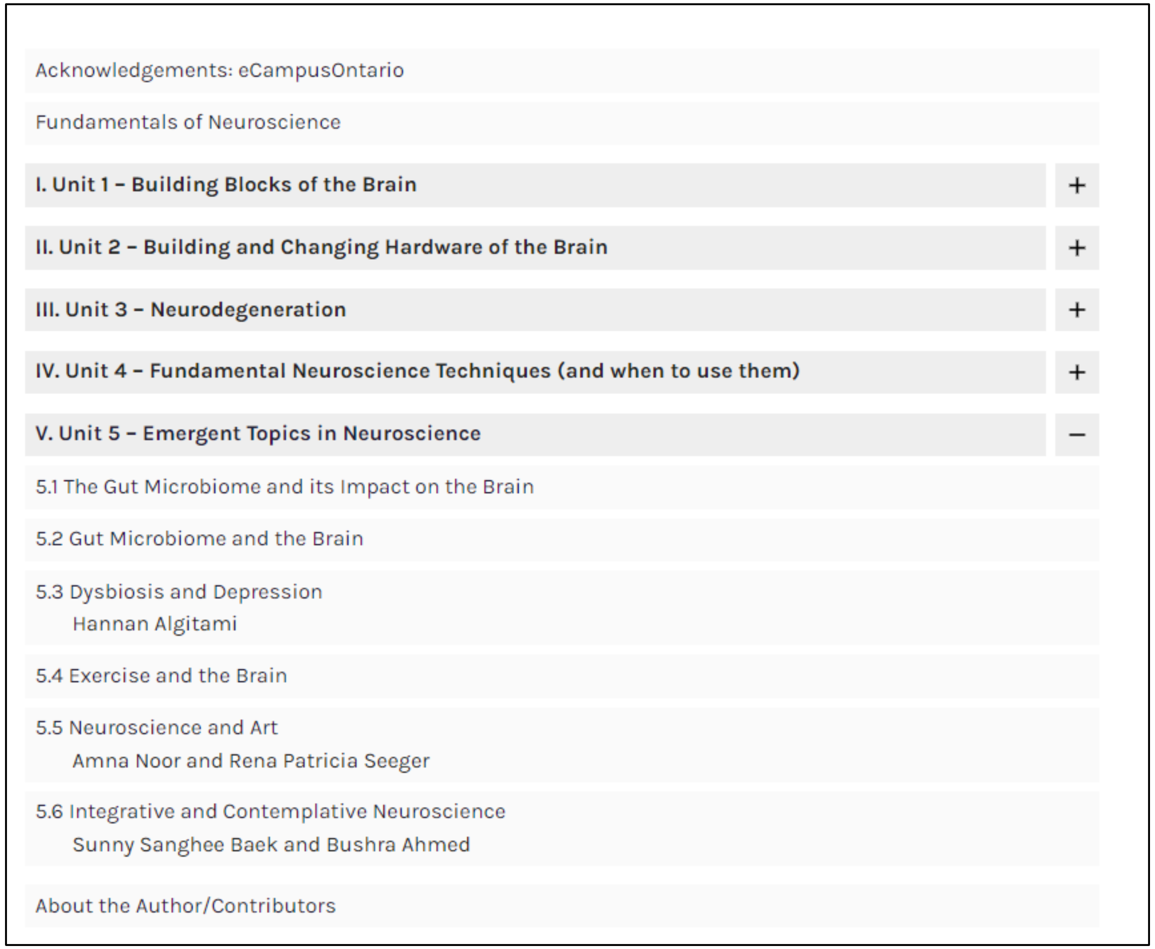

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II. Unit 2 – Building and Changing Hardware of the Brain +

III. Unit 3 – Neurodegeneration +

IV. Unit 4 – Fundamental Neuroscience Techniques (and when to use them) +

V. Unit 5 – Emergent Topics in Neuroscience –

5.1 The Gut Microbiome and its Impact on the Brain

5.2 Gut Microbiome and the Brain

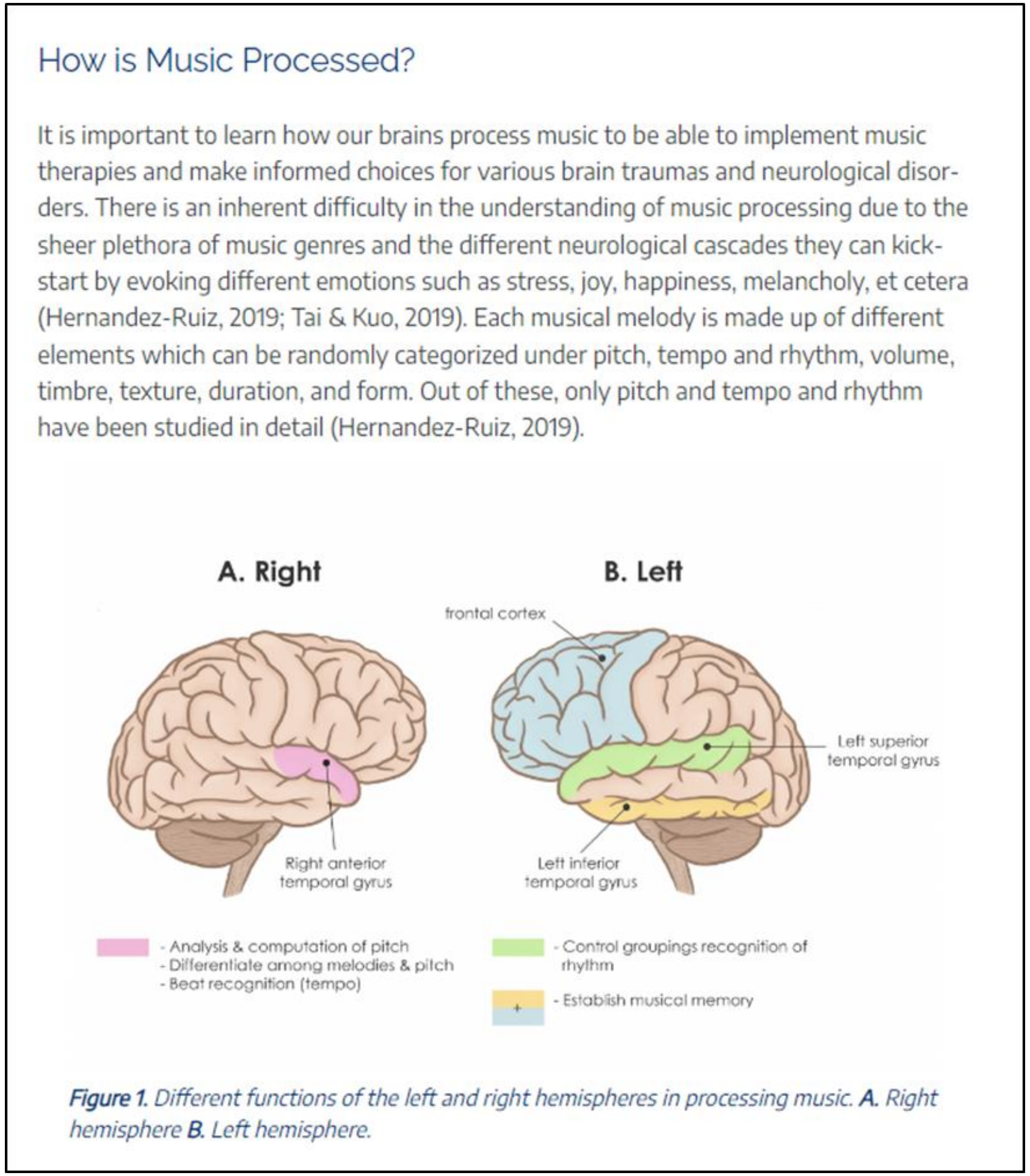
5.3 Dysbiosis and Depression
    Hannan Algitami

5.4 Exercise and the Brain

5.5 Neuroscience and Art
    Amna Noor and Rena Patricia Seeger

5.6 Integrative and Contemplative Neuroscience
    Sunny Sanghee Baek and Bushra Ahmed

About the Author/Contributors



How is Music Processed?

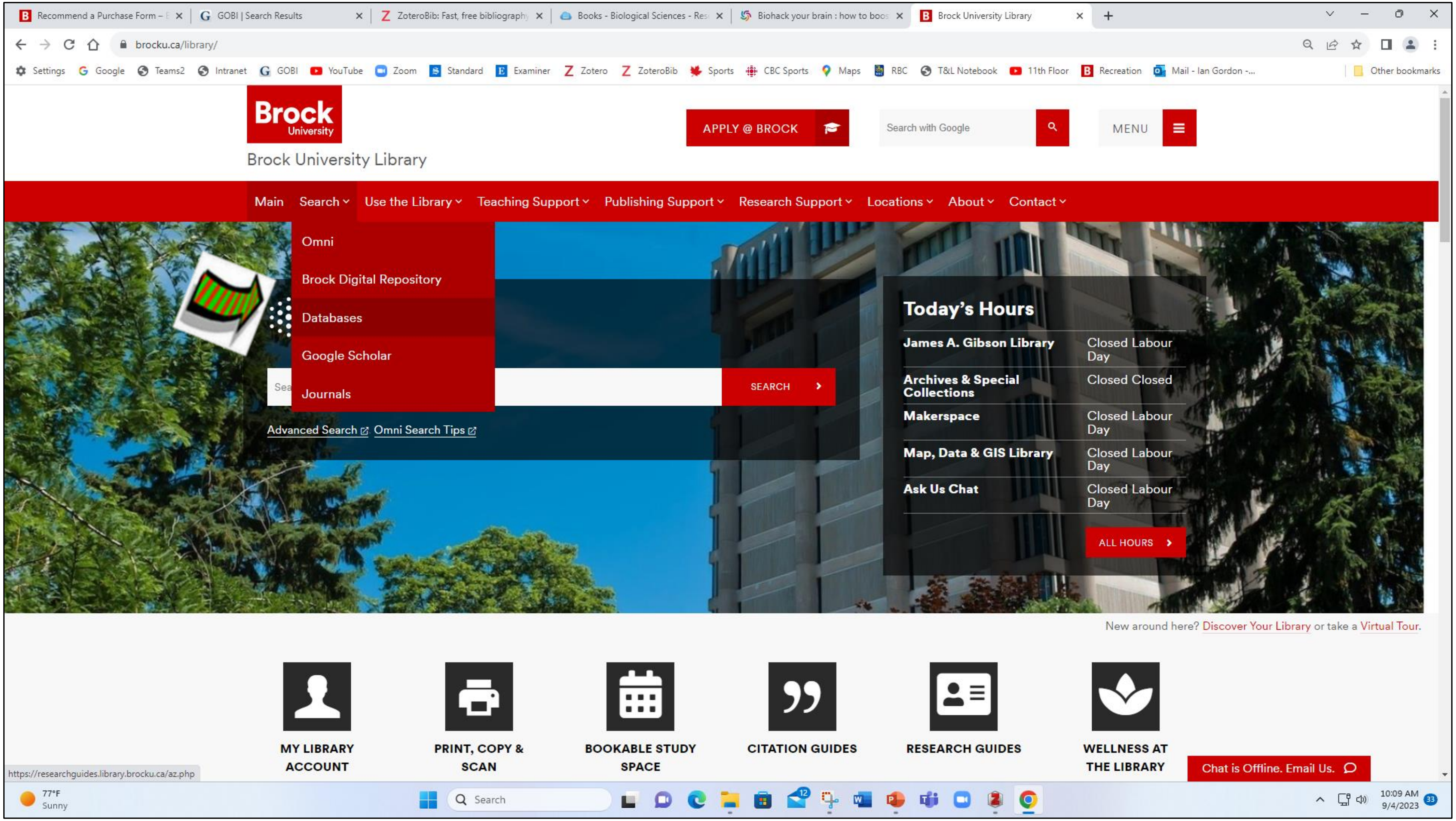
It is important to learn how our brains process music to be able to implement music therapies and make informed choices for various brain traumas and neurological disorders. There is an inherent difficulty in the understanding of music processing due to the sheer plethora of music genres and the different neurological cascades they can kick-start by evoking different emotions such as stress, joy, happiness, melancholy, et cetera (Hernandez-Ruiz, 2019; Tai & Kuo, 2019). Each musical melody is made up of different elements which can be randomly categorized under pitch, tempo and rhythm, volume, timbre, texture, duration, and form. Out of these, only pitch and tempo and rhythm have been studied in detail (Hernandez-Ruiz, 2019).

A. Right
B. Left
frontal cortex
Left superior temporal gyrus
Right anterior temporal gyrus
Left inferior temporal gyrus
🟪 - Analysis & computation of pitch
- Differentiate among melodies & pitch
- Beat recognition (tempo)
🟩 - Control groupings recognition of rhythm
🟨 + 🟦 - Establish musical memory

***Figure 1.** Different functions of the left and right hemispheres in processing music. **A.** Right hemisphere **B.** Left hemisphere.*

Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers



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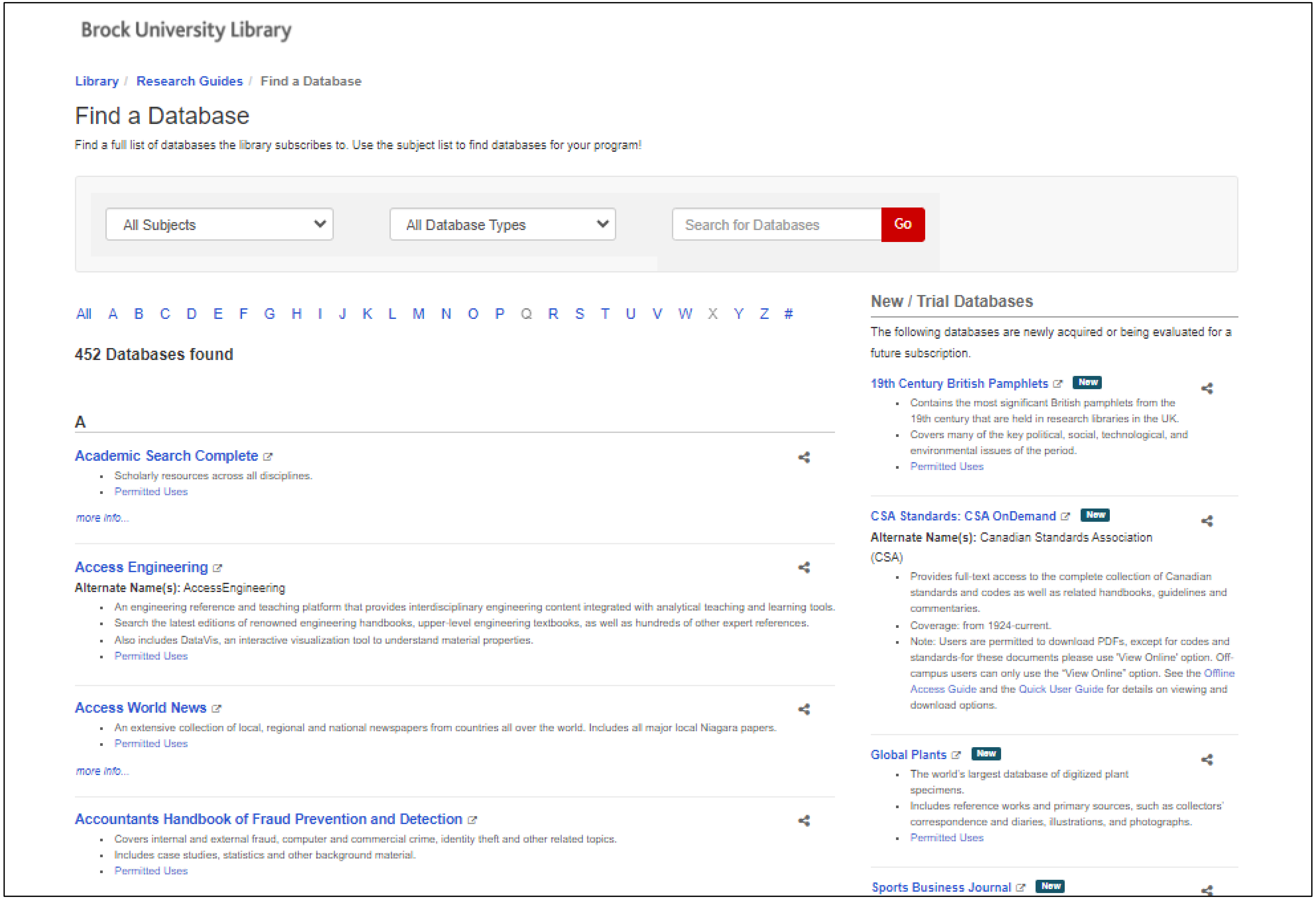
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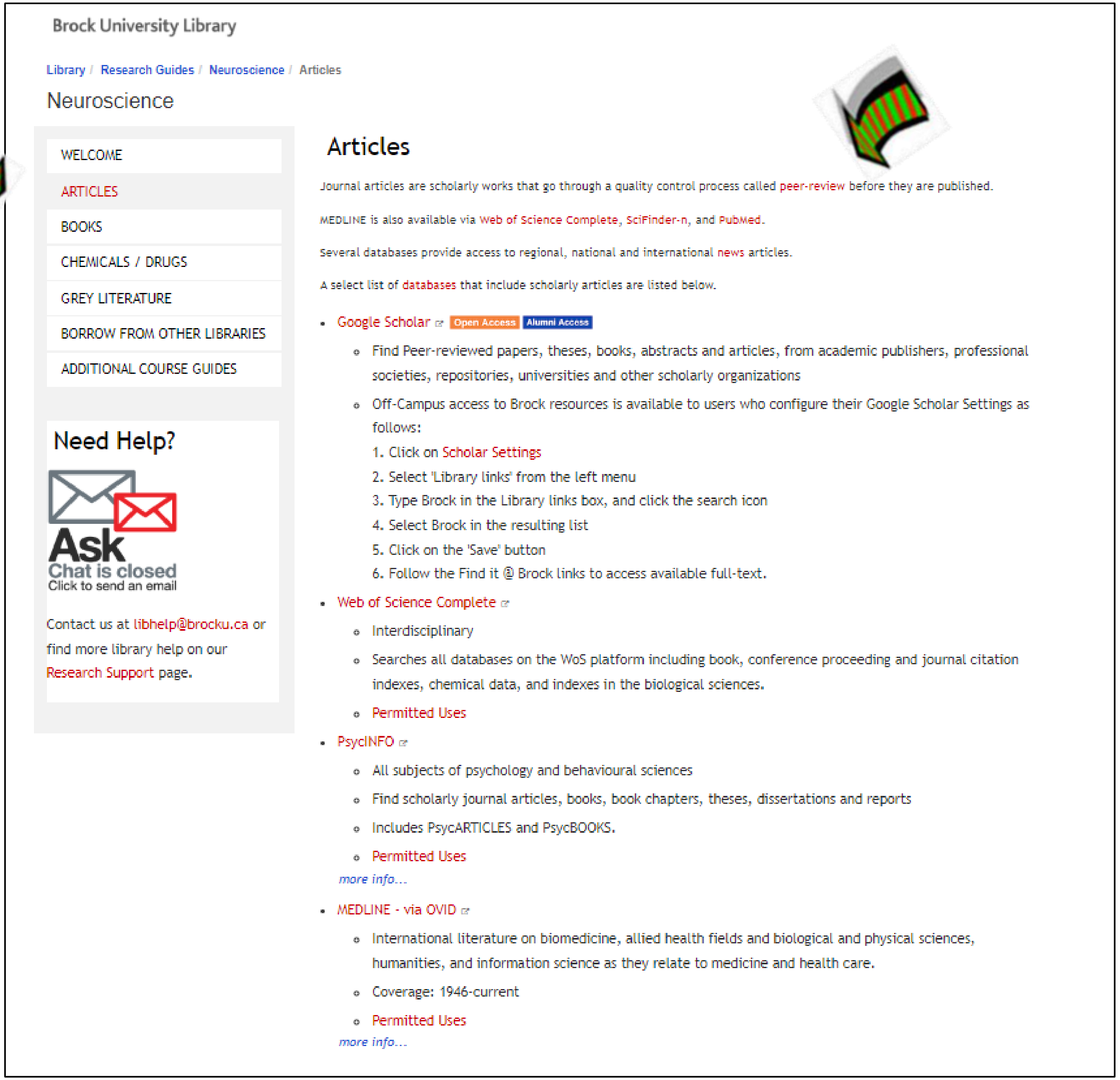

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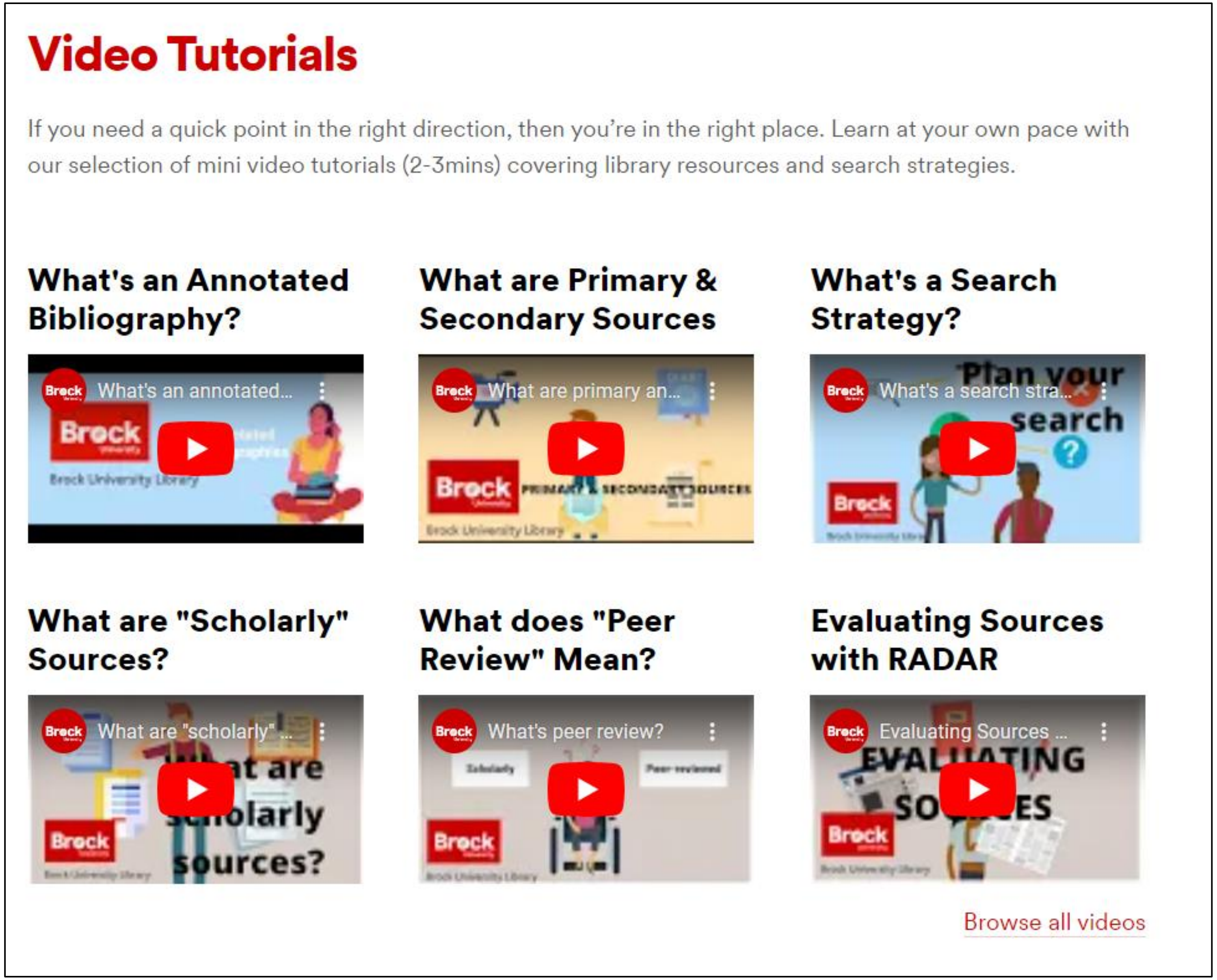

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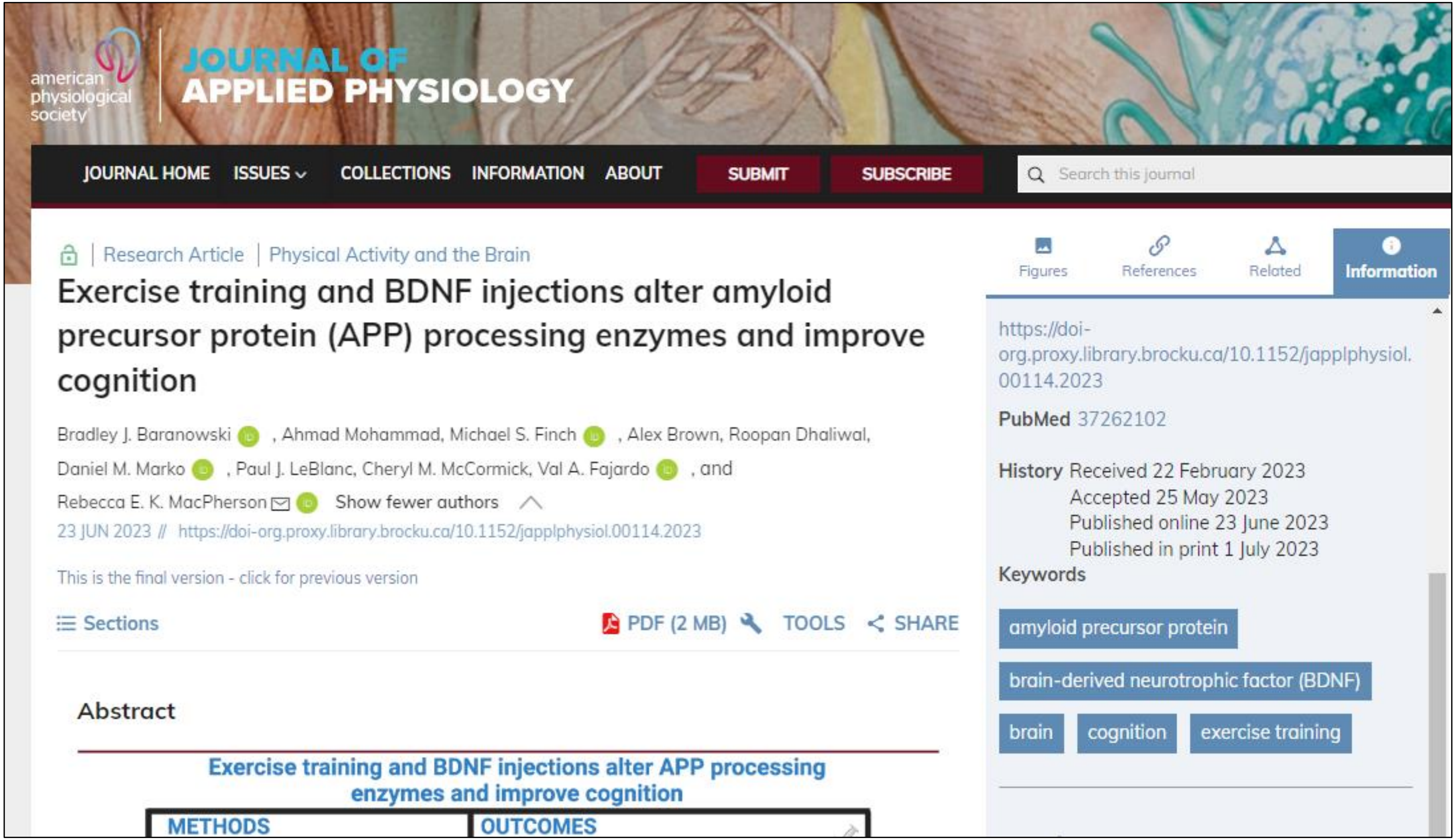
- Where, how and when to get help!
- Questions / Answers



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Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

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Abstract

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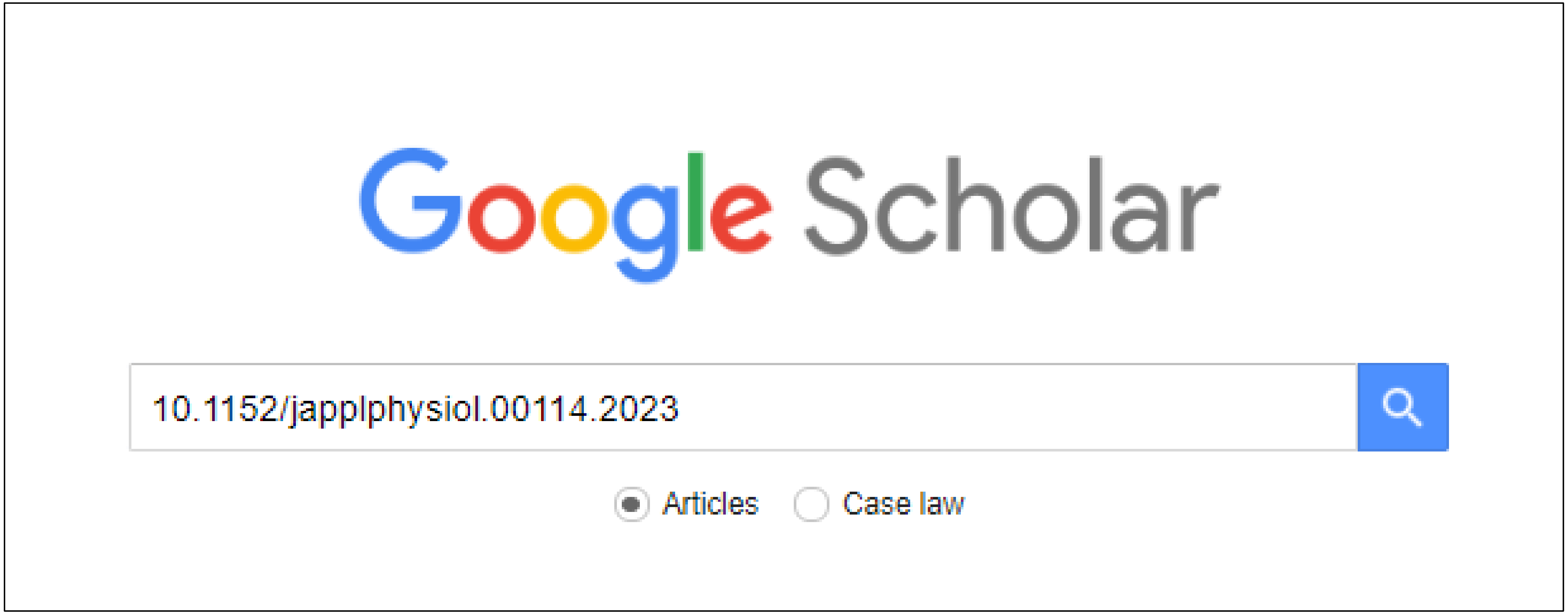

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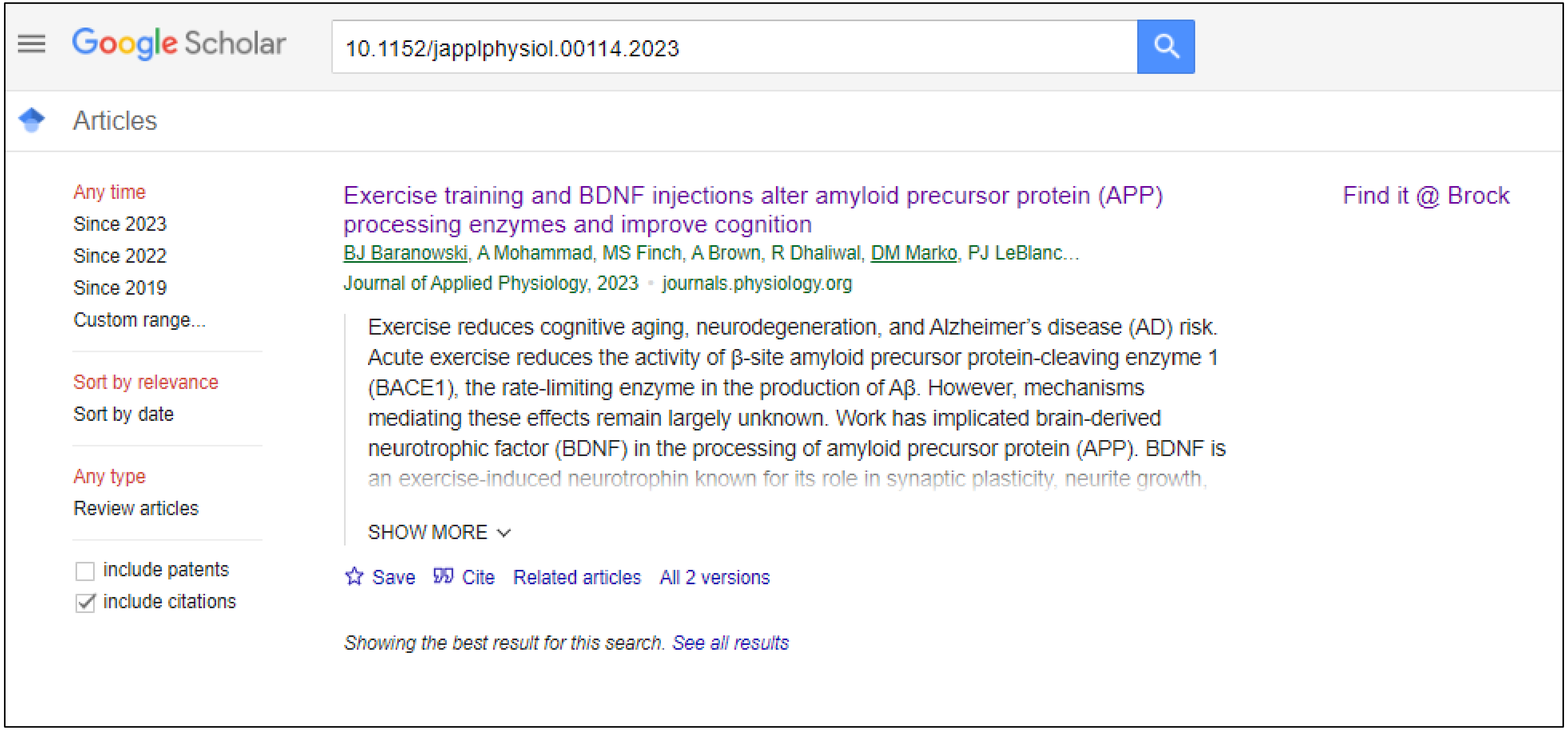

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Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β-site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of Aβ. However, mechanisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth,

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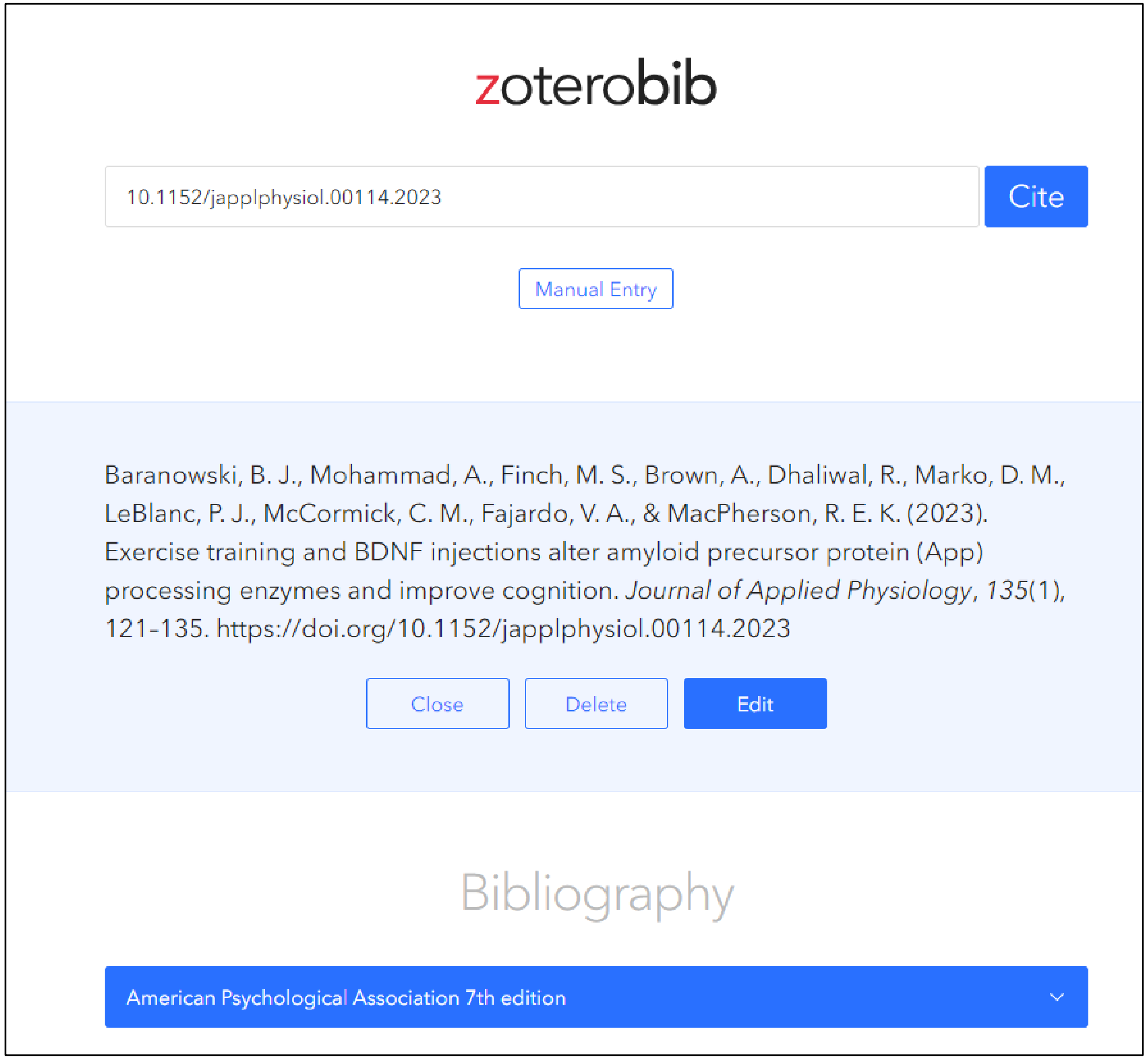

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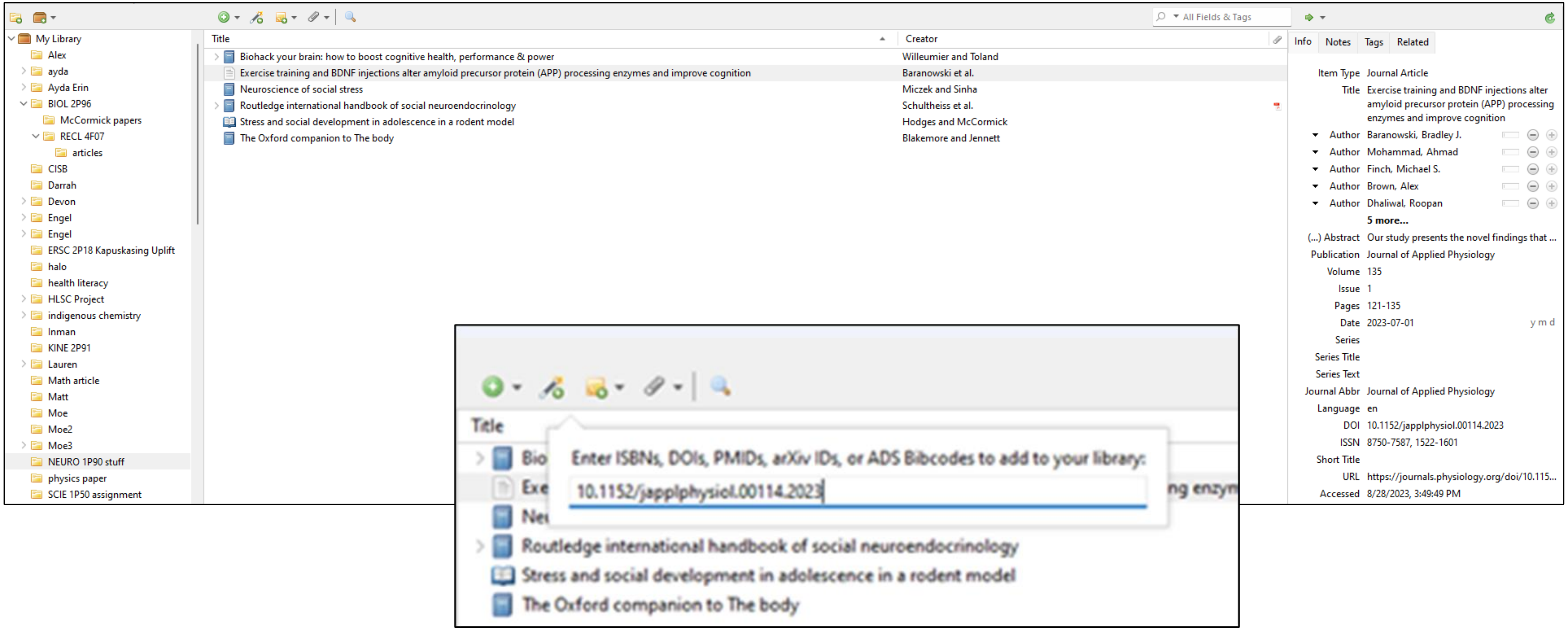

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Author	Finch, Michael S.
Author	Brown, Alex
Author	Dhaliwal, Roopan
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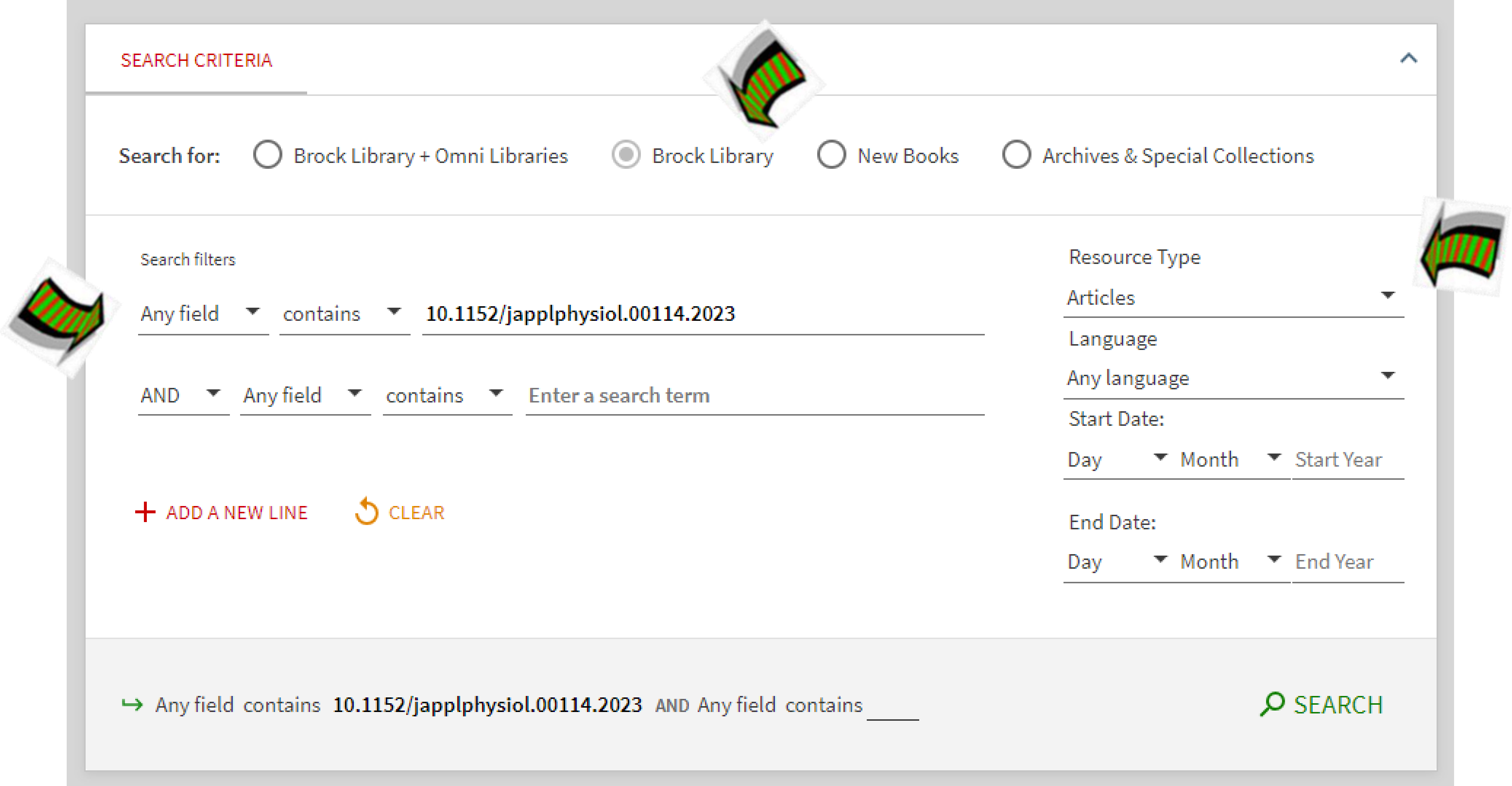

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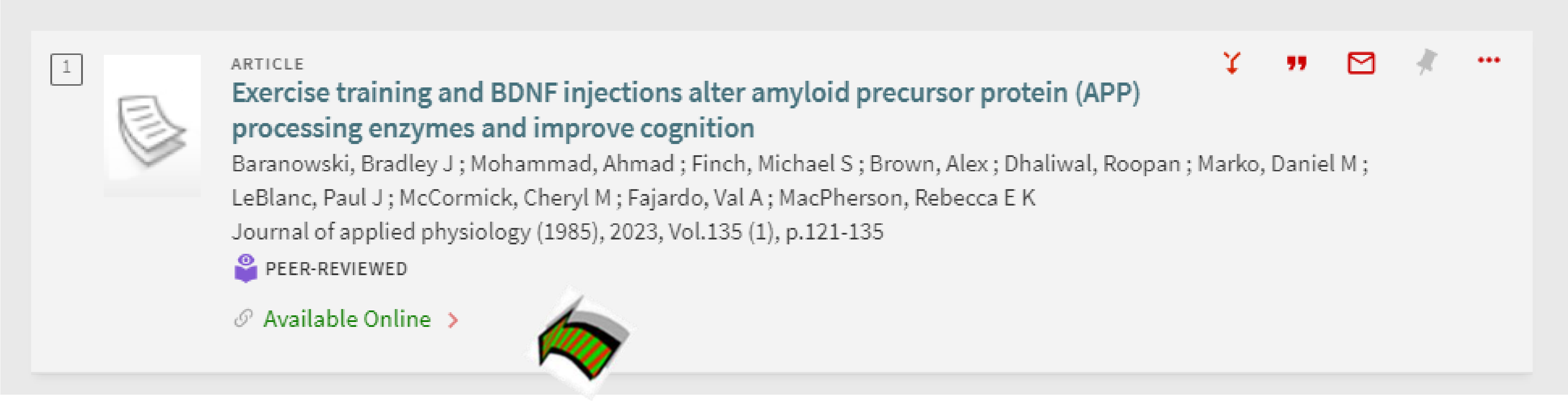

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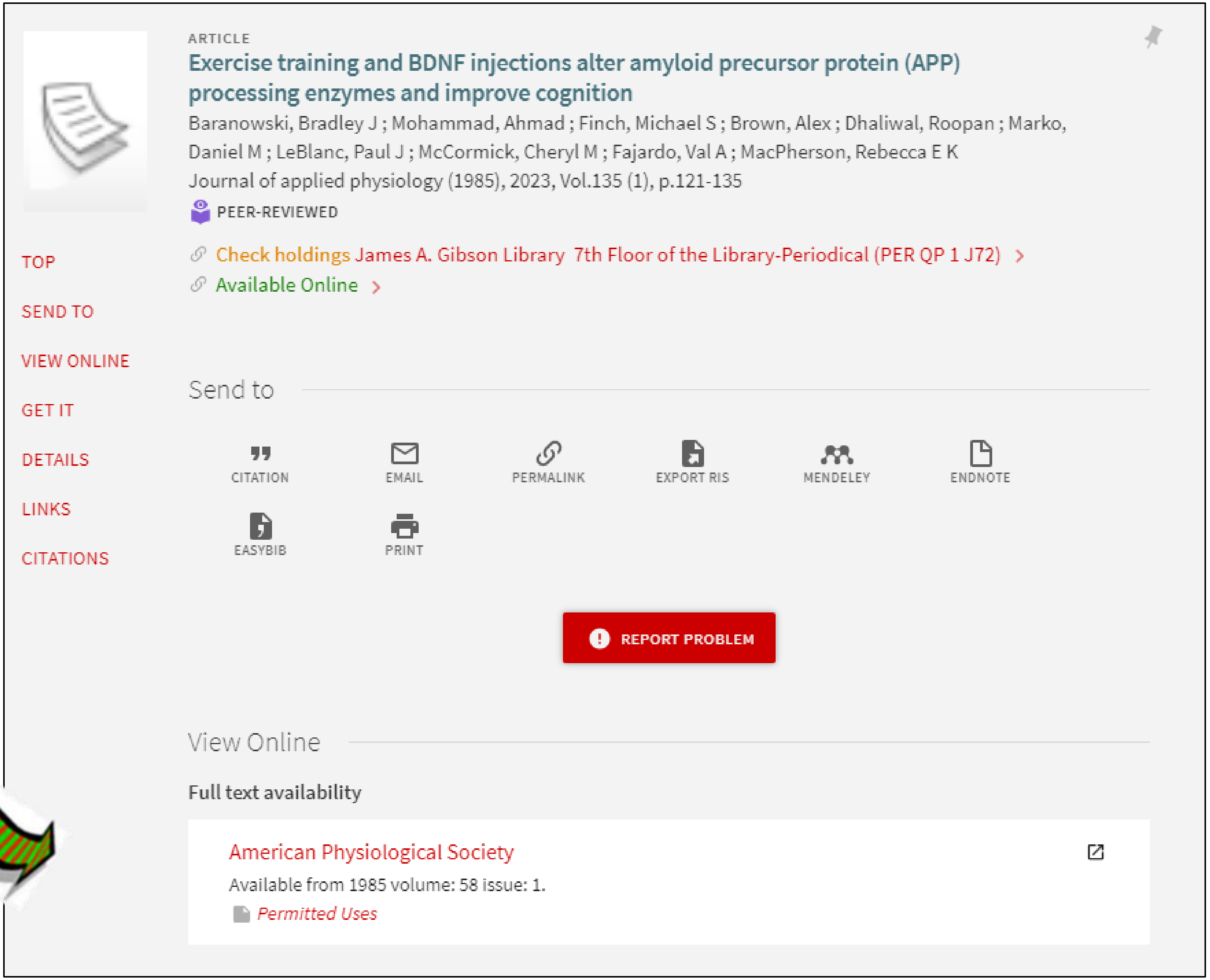

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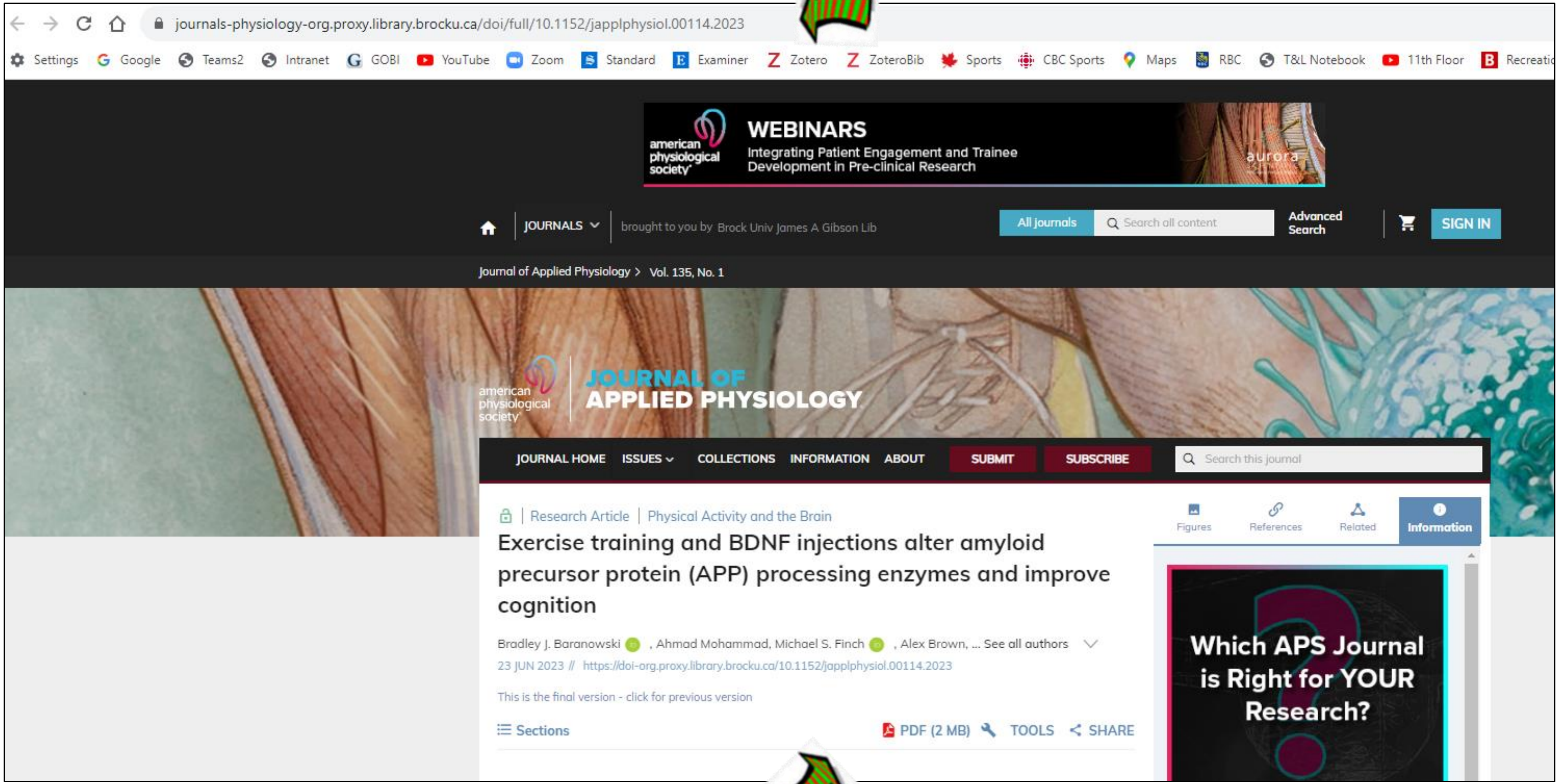

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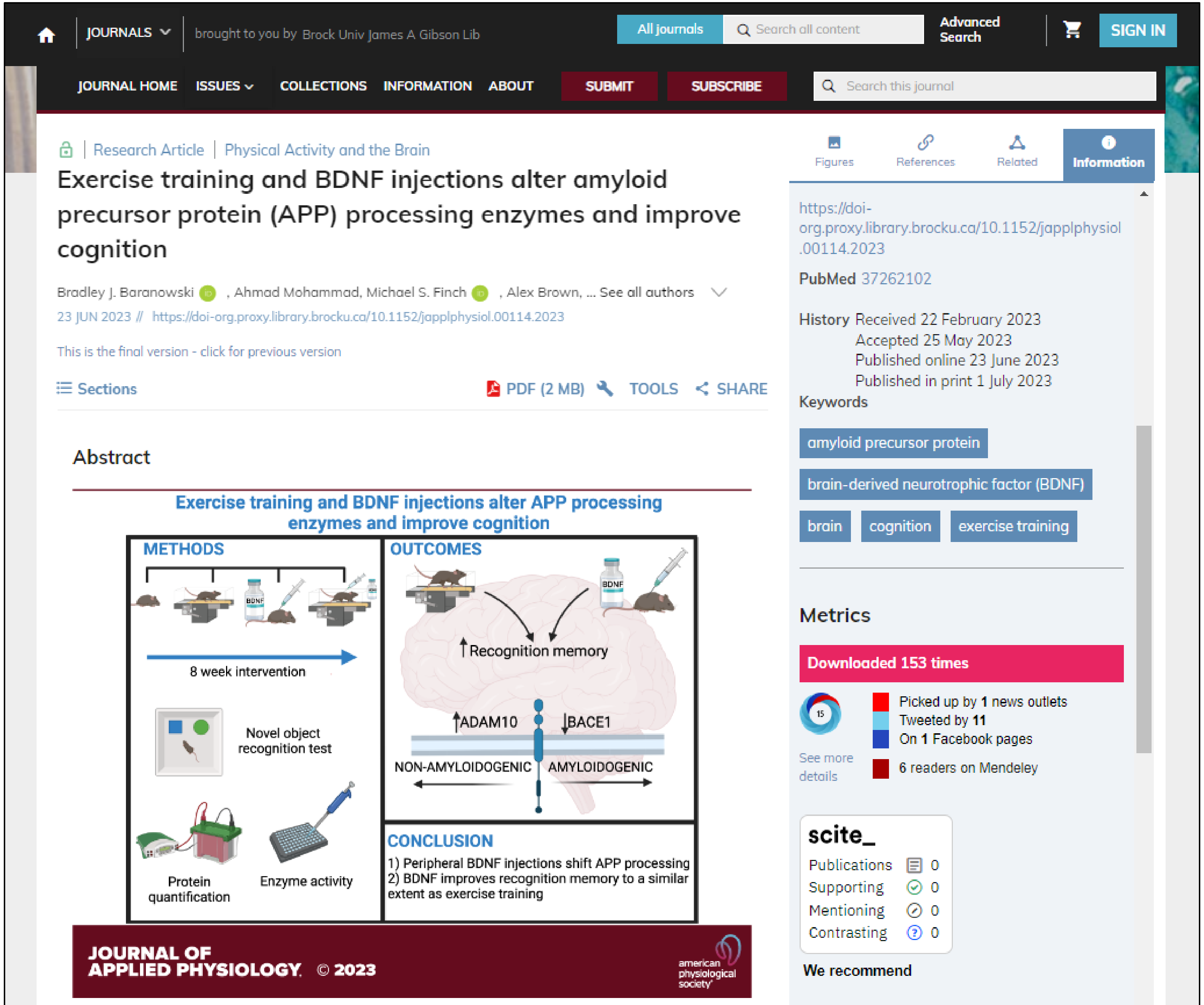

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Abstract

CONCLUSION
1) Peripheral BDNF injections shift APP processing
2) BDNF improves recognition memory to a similar extent as exercise training

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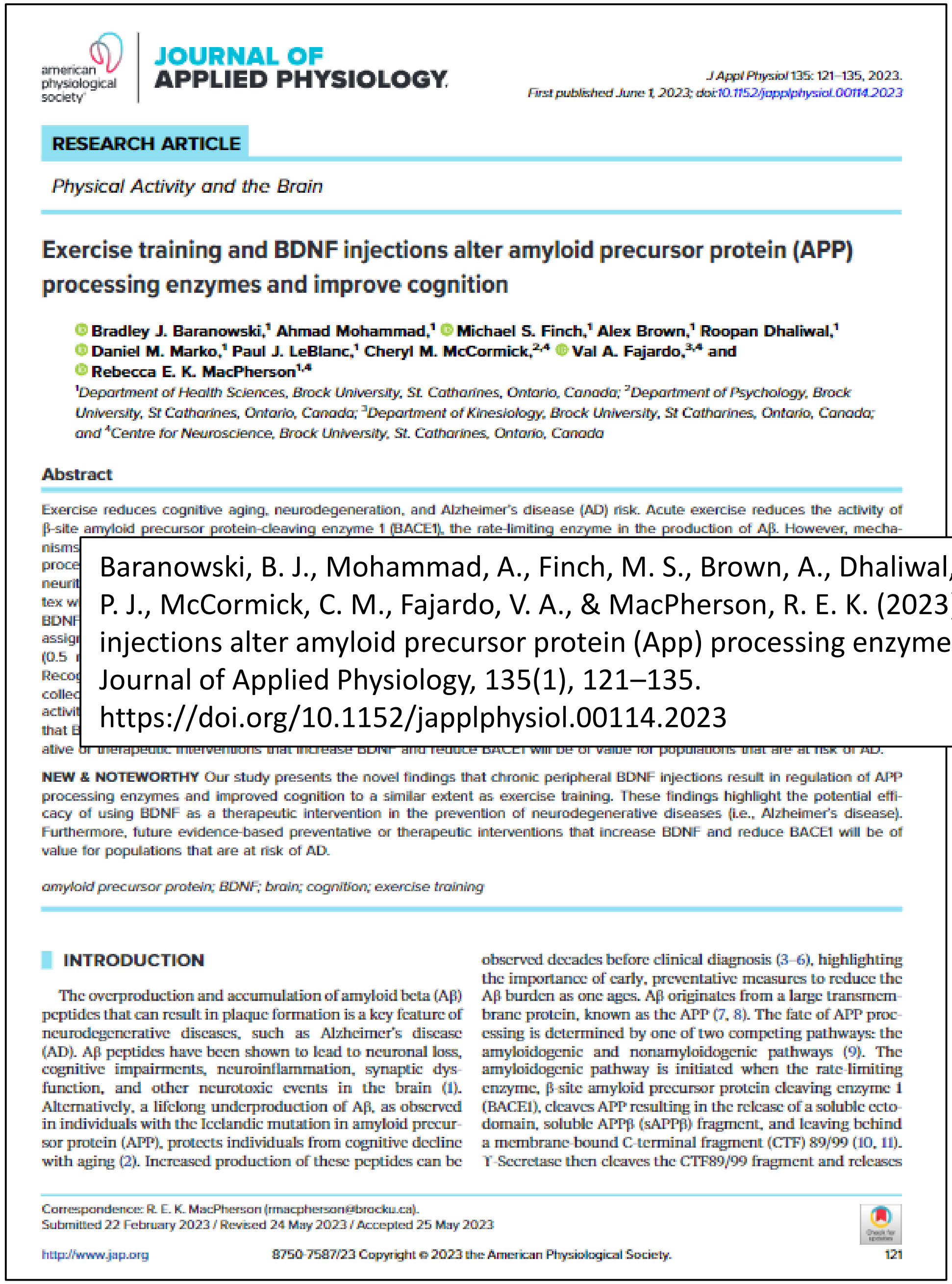

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RESEARCH ARTICLE

Physical Activity and the Brain

Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

Bradley J. Baranowski,[1] Ahmad Mohammad,[1] Michael S. Finch,[1] Alex Brown,[1] Roopan Dhaliwal,[1] Daniel M. Marko,[1] Paul J. LeBlanc,[1] Cheryl M. McCormick,[2,4] Val A. Fajardo,[3,4] and Rebecca E. K. MacPherson[1,4]

[1]Department of Health Sciences, Brock University, St. Catharines, Ontario, Canada; [2]Department of Psychology, Brock University, St Catharines, Ontario, Canada; [3]Department of Kinesiology, Brock University, St Catharines, Ontario, Canada; and [4]Centre for Neuroscience, Brock University, St. Catharines, Ontario, Canada

Abstract

Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β-site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of Aβ. However, mecha-
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NEW & NOTEWORTHY Our study presents the novel findings that chronic peripheral BDNF injections result in regulation of APP processing enzymes and improved cognition to a similar extent as exercise training. These findings highlight the potential efficacy of using BDNF as a therapeutic intervention in the prevention of neurodegenerative diseases (i.e., Alzheimer's disease). Furthermore, future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

amyloid precursor protein; BDNF; brain; cognition; exercise training

INTRODUCTION

The overproduction and accumulation of amyloid beta (Aβ) peptides that can result in plaque formation is a key feature of neurodegenerative diseases, such as Alzheimer's disease (AD). Aβ peptides have been shown to lead to neuronal loss, cognitive impairments, neuroinflammation, synaptic dysfunction, and other neurotoxic events in the brain (1). Alternatively, a lifelong underproduction of Aβ, as observed in individuals with the Icelandic mutation in amyloid precursor protein (APP), protects individuals from cognitive decline with aging (2). Increased production of these peptides can be observed decades before clinical diagnosis (3–6), highlighting the importance of early, preventative measures to reduce the Aβ burden as one ages. Aβ originates from a large transmembrane protein, known as the APP (7, 8). The fate of APP processing is determined by one of two competing pathways: the amyloidogenic and nonamyloidogenic pathways (9). The amyloidogenic pathway is initiated when the rate-limiting enzyme, β-site amyloid precursor protein cleaving enzyme 1 (BACE1), cleaves APP resulting in the release of a soluble ectodomain, soluble APPβ (sAPPβ) fragment, and leaving behind a membrane-bound C-terminal fragment (CTF) 89/99 (10, 11). Ɣ-Secretase then cleaves the CTF89/99 fragment and releases

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Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β-site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of Aβ. However, mechanisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth, and neuronal survival. Previously, our lab has shown using an ex vivo model that treatment of the prefrontal cortex with BDNF reduced BACE1 activity, highlighting a BDNF to BACE1 link. The purpose of this research was to examine whether BDNF treatments resulted in similar biochemical adaptations to APP processing as exercise training. Male C57BL6/J mice were assigned into one of four groups (*n* = 12/group): *1*) control; *2*) exercise training (progressive treadmill training 5 days/wk); *3*) BDNF (0.5 mg/kg body mass subcutaneous injection 5 days/wk); or *4*) endurance training and BDNF, for an 8-wk intervention. Recognition memory was measured with a novel object recognition test. Serum, the prefrontal cortex, and hippocampus were collected. BDNF improved recognition memory to a similar extent as endurance training. BDNF and exercise decreased BACE1 activity and increased ADAM10 activity in the prefrontal cortex, indicating a shift in APP processing. Our novel results indicate that BDNF exerts similar beneficial effects on cognition and APP processing as exercise training. Future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

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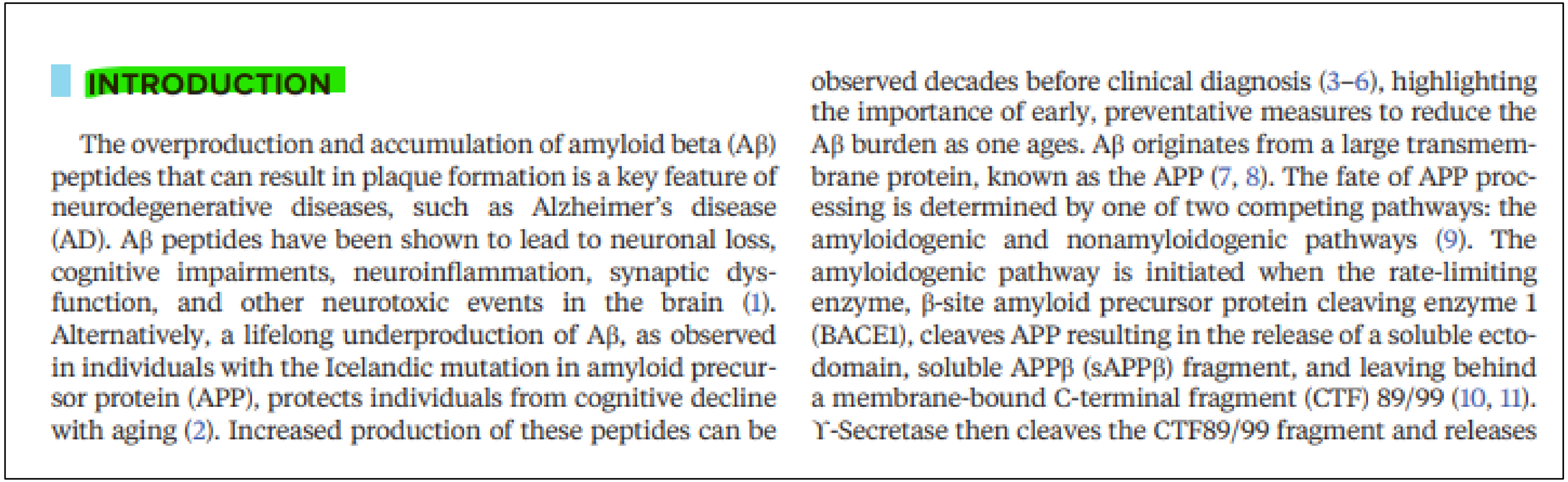

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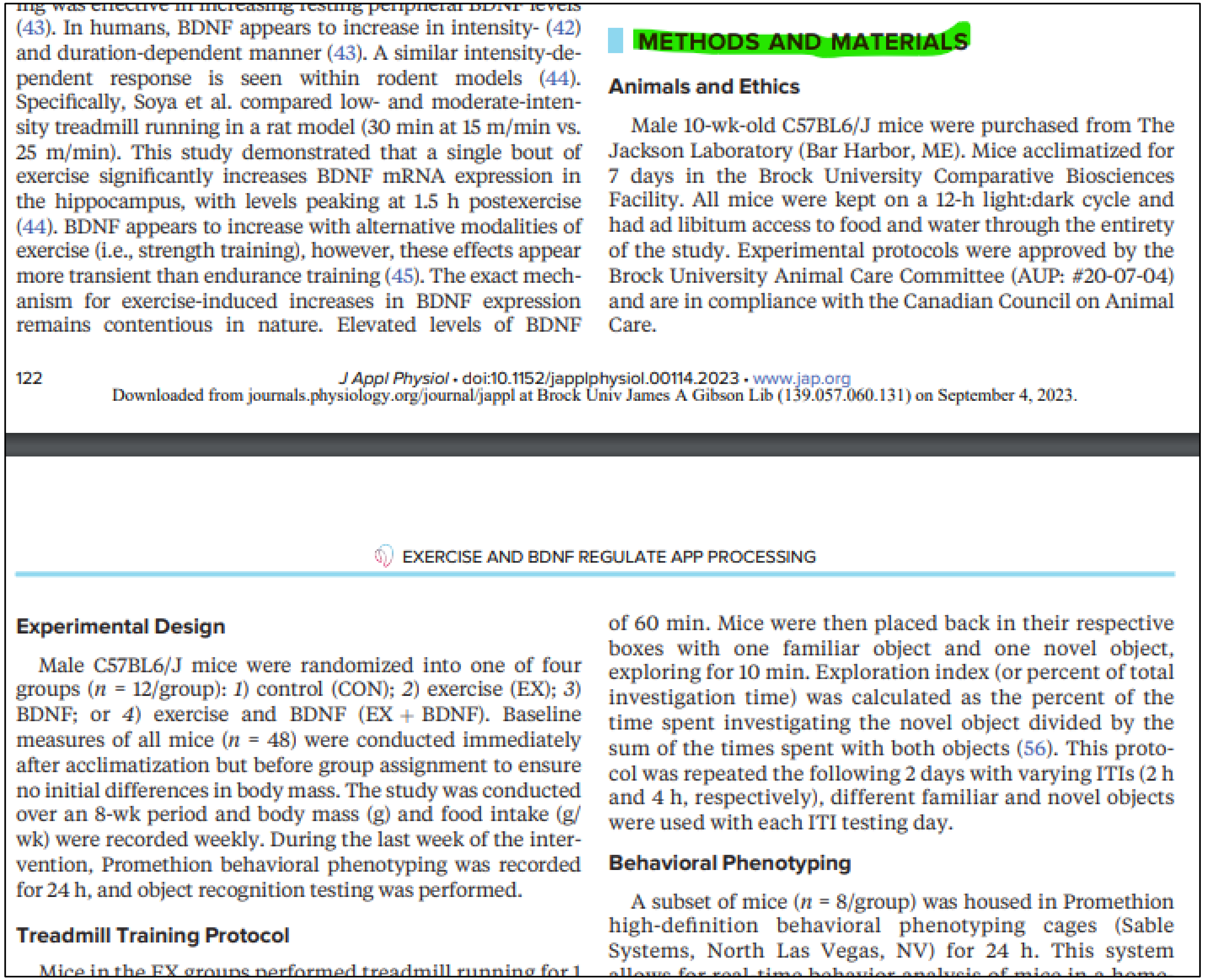

ing was effective in increasing resting peripheral BDNF levels (43). In humans, BDNF appears to increase in intensity- (42) and duration-dependent manner (43). A similar intensity-dependent response is seen within rodent models (44). Specifically, Soya et al. compared low- and moderate-intensity treadmill running in a rat model (30 min at 15 m/min vs. 25 m/min). This study demonstrated that a single bout of exercise significantly increases BDNF mRNA expression in the hippocampus, with levels peaking at 1.5 h postexercise (44). BDNF appears to increase with alternative modalities of exercise (i.e., strength training), however, these effects appear more transient than endurance training (45). The exact mechanism for exercise-induced increases in BDNF expression remains contentious in nature. Elevated levels of BDNF

METHODS AND MATERIALS

Animals and Ethics

Male 10-wk-old C57BL6/J mice were purchased from The Jackson Laboratory (Bar Harbor, ME). Mice acclimatized for 7 days in the Brock University Comparative Biosciences Facility. All mice were kept on a 12-h light:dark cycle and had ad libitum access to food and water through the entirety of the study. Experimental protocols were approved by the Brock University Animal Care Committee (AUP: #20-07-04) and are in compliance with the Canadian Council on Animal Care.

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Experimental Design

Male C57BL6/J mice were randomized into one of four groups (n = 12/group): *1*) control (CON); *2*) exercise (EX); *3*) BDNF; or *4*) exercise and BDNF (EX + BDNF). Baseline measures of all mice (n = 48) were conducted immediately after acclimatization but before group assignment to ensure no initial differences in body mass. The study was conducted over an 8-wk period and body mass (g) and food intake (g/wk) were recorded weekly. During the last week of the intervention, Promethion behavioral phenotyping was recorded for 24 h, and object recognition testing was performed.

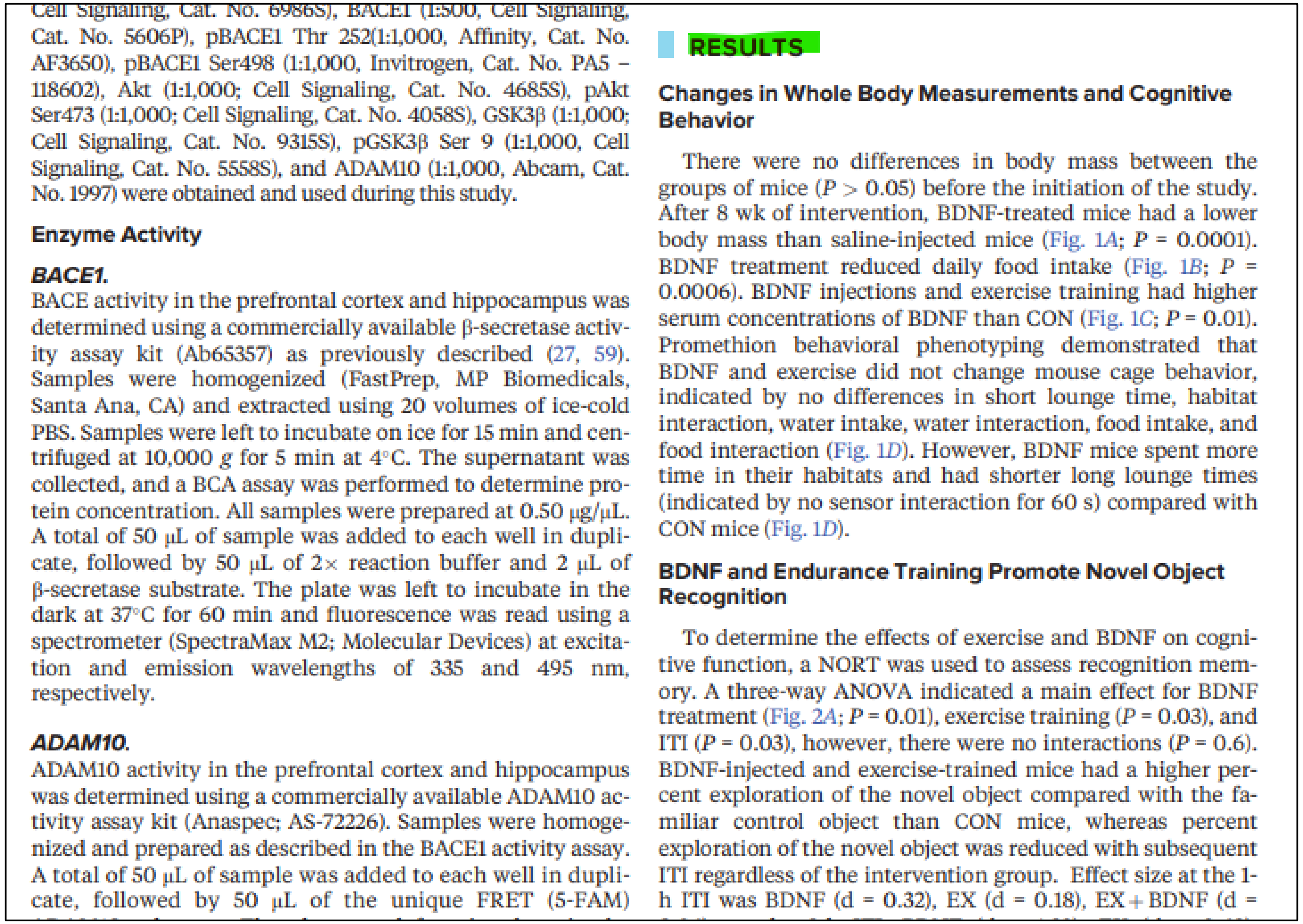
Treadmill Training Protocol

Mice in the EX groups performed treadmill running for 1

of 60 min. Mice were then placed back in their respective boxes with one familiar object and one novel object, exploring for 10 min. Exploration index (or percent of total investigation time) was calculated as the percent of the time spent investigating the novel object divided by the sum of the times spent with both objects (56). This protocol was repeated the following 2 days with varying ITIs (2 h and 4 h, respectively), different familiar and novel objects were used with each ITI testing day.

Behavioral Phenotyping

A subset of mice (n = 8/group) was housed in Promethion high-definition behavioral phenotyping cages (Sable Systems, North Las Vegas, NV) for 24 h. This system allows for real-time behavior analysis of mice in a home-



Cell Signaling, Cat. No. 6986S), BACE1 (1:500, Cell Signaling, Cat. No. 5606P), pBACE1 Thr 252(1:1,000, Affinity, Cat. No. AF3650), pBACE1 Ser498 (1:1,000, Invitrogen, Cat. No. PA5 – 118602), Akt (1:1,000; Cell Signaling, Cat. No. 4685S), pAkt Ser473 (1:1,000; Cell Signaling, Cat. No. 4058S), GSK3β (1:1,000; Cell Signaling, Cat. No. 9315S), pGSK3β Ser 9 (1:1,000, Cell Signaling, Cat. No. 5558S), and ADAM10 (1:1,000, Abcam, Cat. No. 1997) were obtained and used during this study.

Enzyme Activity

BACE1.

BACE activity in the prefrontal cortex and hippocampus was determined using a commercially available β-secretase activity assay kit (Ab65357) as previously described (27, 59). Samples were homogenized (FastPrep, MP Biomedicals, Santa Ana, CA) and extracted using 20 volumes of ice-cold PBS. Samples were left to incubate on ice for 15 min and centrifuged at 10,000 *g* for 5 min at 4°C. The supernatant was collected, and a BCA assay was performed to determine protein concentration. All samples were prepared at 0.50 μg/μL. A total of 50 μL of sample was added to each well in duplicate, followed by 50 μL of 2× reaction buffer and 2 μL of β-secretase substrate. The plate was left to incubate in the dark at 37°C for 60 min and fluorescence was read using a spectrometer (SpectraMax M2; Molecular Devices) at excitation and emission wavelengths of 335 and 495 nm, respectively.

ADAM10.

ADAM10 activity in the prefrontal cortex and hippocampus was determined using a commercially available ADAM10 activity assay kit (Anaspec; AS-72226). Samples were homogenized and prepared as described in the BACE1 activity assay. A total of 50 μL of sample was added to each well in duplicate, followed by 50 μL of the unique FRET (5-FAM)

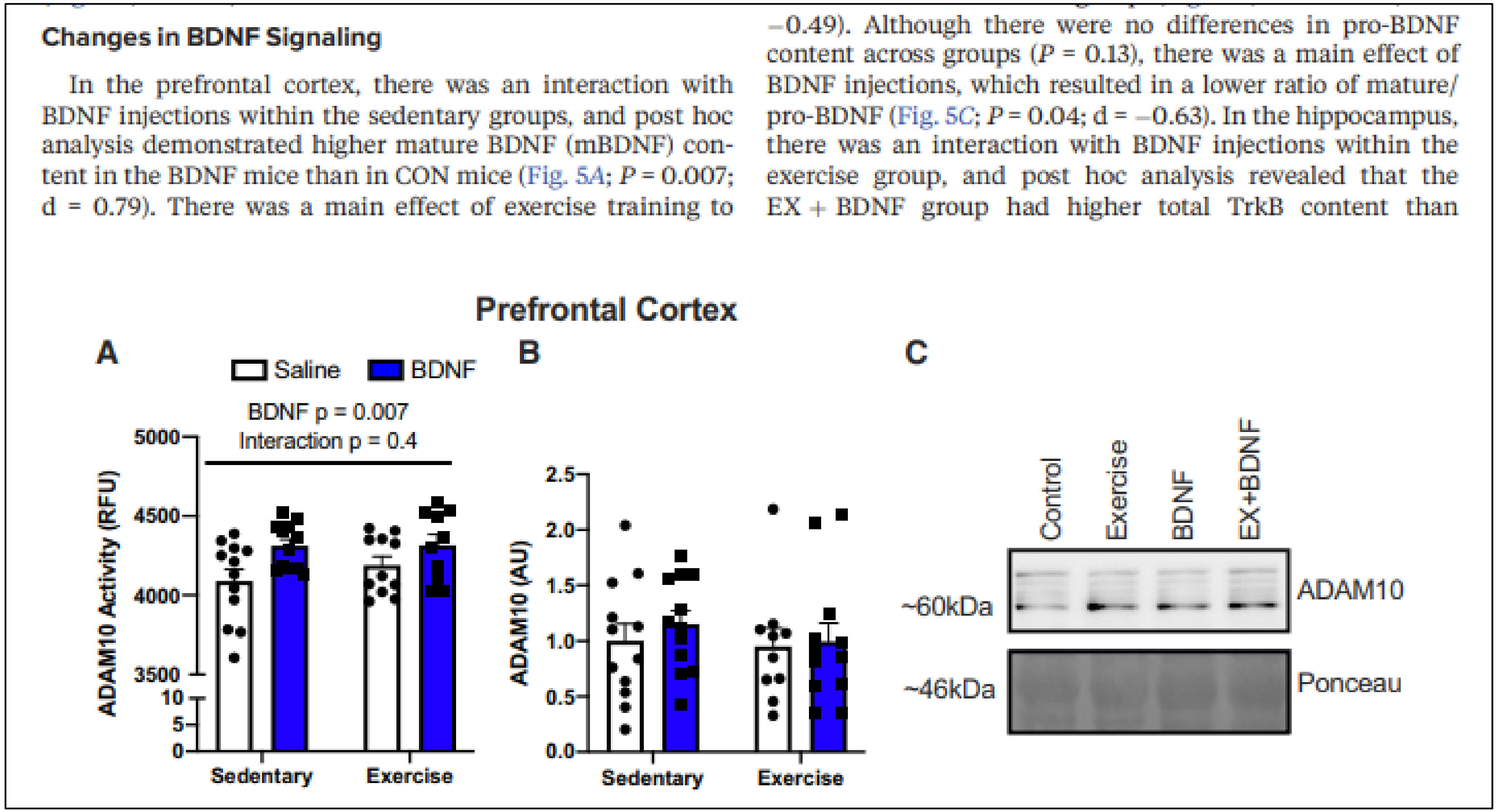
RESULTS

Changes in Whole Body Measurements and Cognitive Behavior

There were no differences in body mass between the groups of mice ($P > 0.05$) before the initiation of the study. After 8 wk of intervention, BDNF-treated mice had a lower body mass than saline-injected mice (Fig. 1*A*; $P = 0.0001$). BDNF treatment reduced daily food intake (Fig. 1*B*; $P = 0.0006$). BDNF injections and exercise training had higher serum concentrations of BDNF than CON (Fig. 1*C*; $P = 0.01$). Promethion behavioral phenotyping demonstrated that BDNF and exercise did not change mouse cage behavior, indicated by no differences in short lounge time, habitat interaction, water intake, water interaction, food intake, and food interaction (Fig. 1*D*). However, BDNF mice spent more time in their habitats and had shorter long lounge times (indicated by no sensor interaction for 60 s) compared with CON mice (Fig. 1*D*).

BDNF and Endurance Training Promote Novel Object Recognition

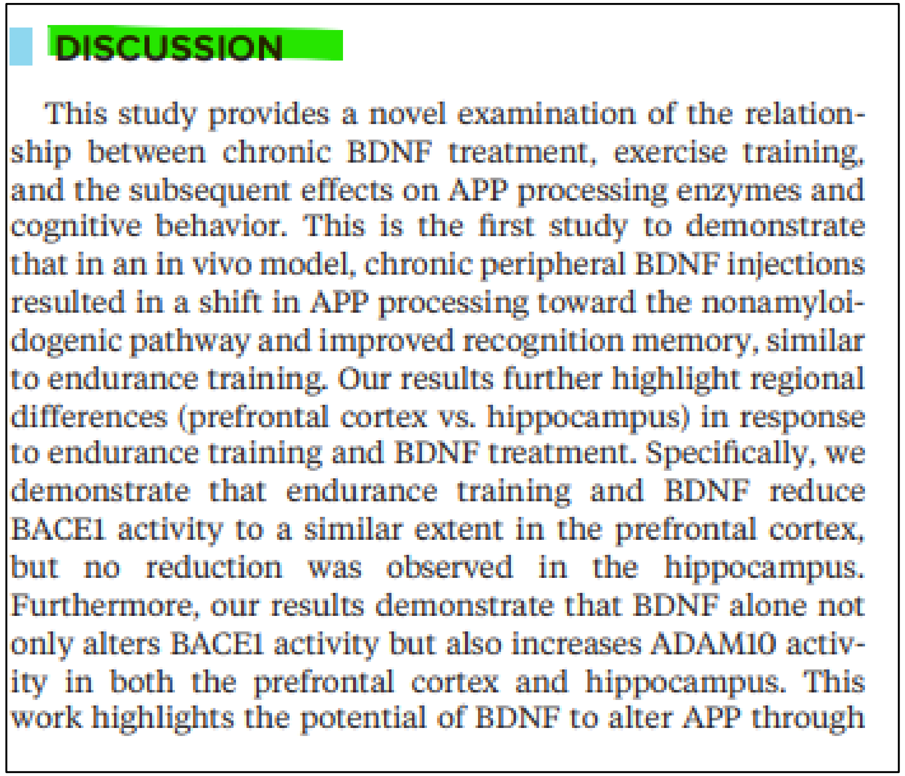
To determine the effects of exercise and BDNF on cognitive function, a NORT was used to assess recognition memory. A three-way ANOVA indicated a main effect for BDNF treatment (Fig. 2*A*; $P = 0.01$), exercise training ($P = 0.03$), and ITI ($P = 0.03$), however, there were no interactions ($P = 0.6$). BDNF-injected and exercise-trained mice had a higher percent exploration of the novel object compared with the familiar control object than CON mice, whereas percent exploration of the novel object was reduced with subsequent ITI regardless of the intervention group. Effect size at the 1-h ITI was BDNF ($d = 0.32$), EX ($d = 0.18$), EX + BDNF ($d =$



Changes in BDNF Signaling

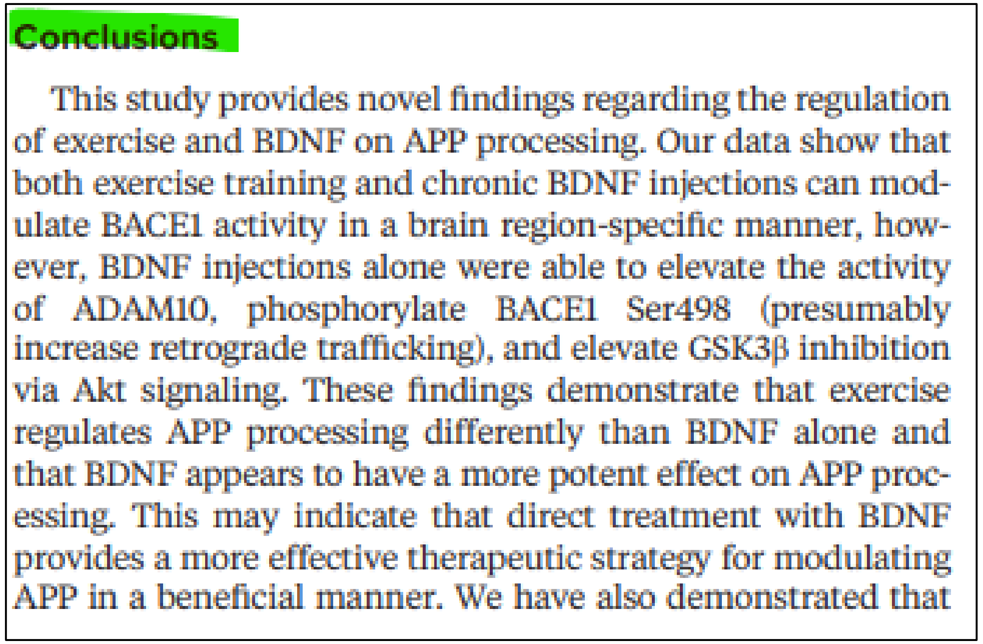
In the prefrontal cortex, there was an interaction with BDNF injections within the sedentary groups, and post hoc analysis demonstrated higher mature BDNF (mBDNF) content in the BDNF mice than in CON mice (Fig. 5*A*; $P = 0.007$; d = 0.79). There was a main effect of exercise training to

–0.49). Although there were no differences in pro-BDNF content across groups ($P = 0.13$), there was a main effect of BDNF injections, which resulted in a lower ratio of mature/pro-BDNF (Fig. 5*C*; $P = 0.04$; d = –0.63). In the hippocampus, there was an interaction with BDNF injections within the exercise group, and post hoc analysis revealed that the EX + BDNF group had higher total TrkB content than



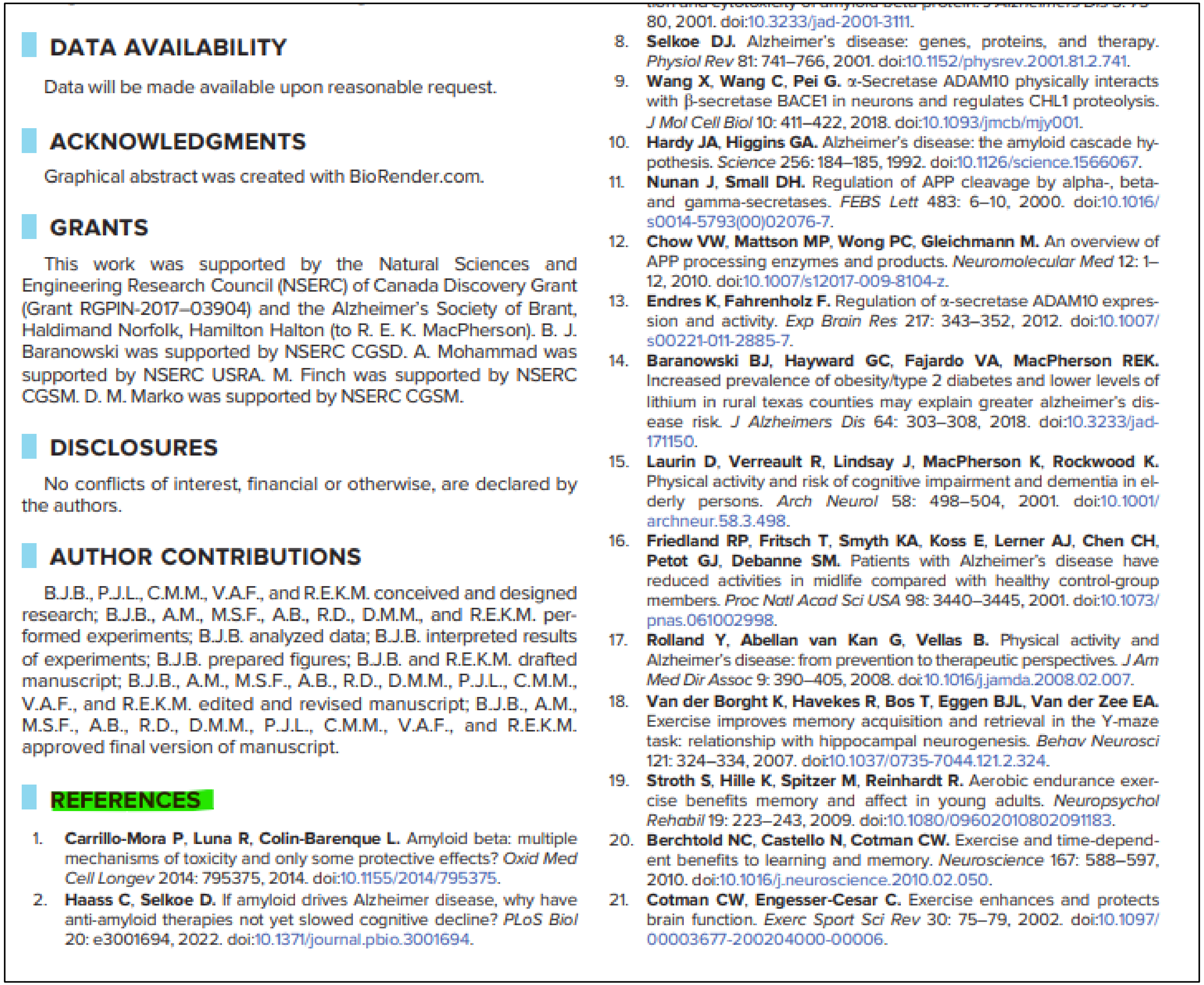
DISCUSSION

This study provides a novel examination of the relationship between chronic BDNF treatment, exercise training, and the subsequent effects on APP processing enzymes and cognitive behavior. This is the first study to demonstrate that in an in vivo model, chronic peripheral BDNF injections resulted in a shift in APP processing toward the nonamyloidogenic pathway and improved recognition memory, similar to endurance training. Our results further highlight regional differences (prefrontal cortex vs. hippocampus) in response to endurance training and BDNF treatment. Specifically, we demonstrate that endurance training and BDNF reduce BACE1 activity to a similar extent in the prefrontal cortex, but no reduction was observed in the hippocampus. Furthermore, our results demonstrate that BDNF alone not only alters BACE1 activity but also increases ADAM10 activity in both the prefrontal cortex and hippocampus. This work highlights the potential of BDNF to alter APP through



Conclusions

This study provides novel findings regarding the regulation of exercise and BDNF on APP processing. Our data show that both exercise training and chronic BDNF injections can modulate BACE1 activity in a brain region-specific manner, however, BDNF injections alone were able to elevate the activity of ADAM10, phosphorylate BACE1 Ser498 (presumably increase retrograde trafficking), and elevate GSK3β inhibition via Akt signaling. These findings demonstrate that exercise regulates APP processing differently than BDNF alone and that BDNF appears to have a more potent effect on APP processing. This may indicate that direct treatment with BDNF provides a more effective therapeutic strategy for modulating APP in a beneficial manner. We have also demonstrated that



DATA AVAILABILITY

Data will be made available upon reasonable request.

ACKNOWLEDGMENTS

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GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

B.J.B., P.J.L., C.M.M., V.A.F., and R.E.K.M. conceived and designed research; B.J.B., A.M., M.S.F., A.B., R.D., D.M.M., and R.E.K.M. performed experiments; B.J.B. analyzed data; B.J.B. interpreted results of experiments; B.J.B. prepared figures; B.J.B. and R.E.K.M. drafted manuscript; B.J.B., A.M., M.S.F., A.B., R.D., D.M.M., P.J.L., C.M.M., V.A.F., and R.E.K.M. edited and revised manuscript; B.J.B., A.M., M.S.F., A.B., R.D., D.M.M., P.J.L., C.M.M., V.A.F., and R.E.K.M. approved final version of manuscript.

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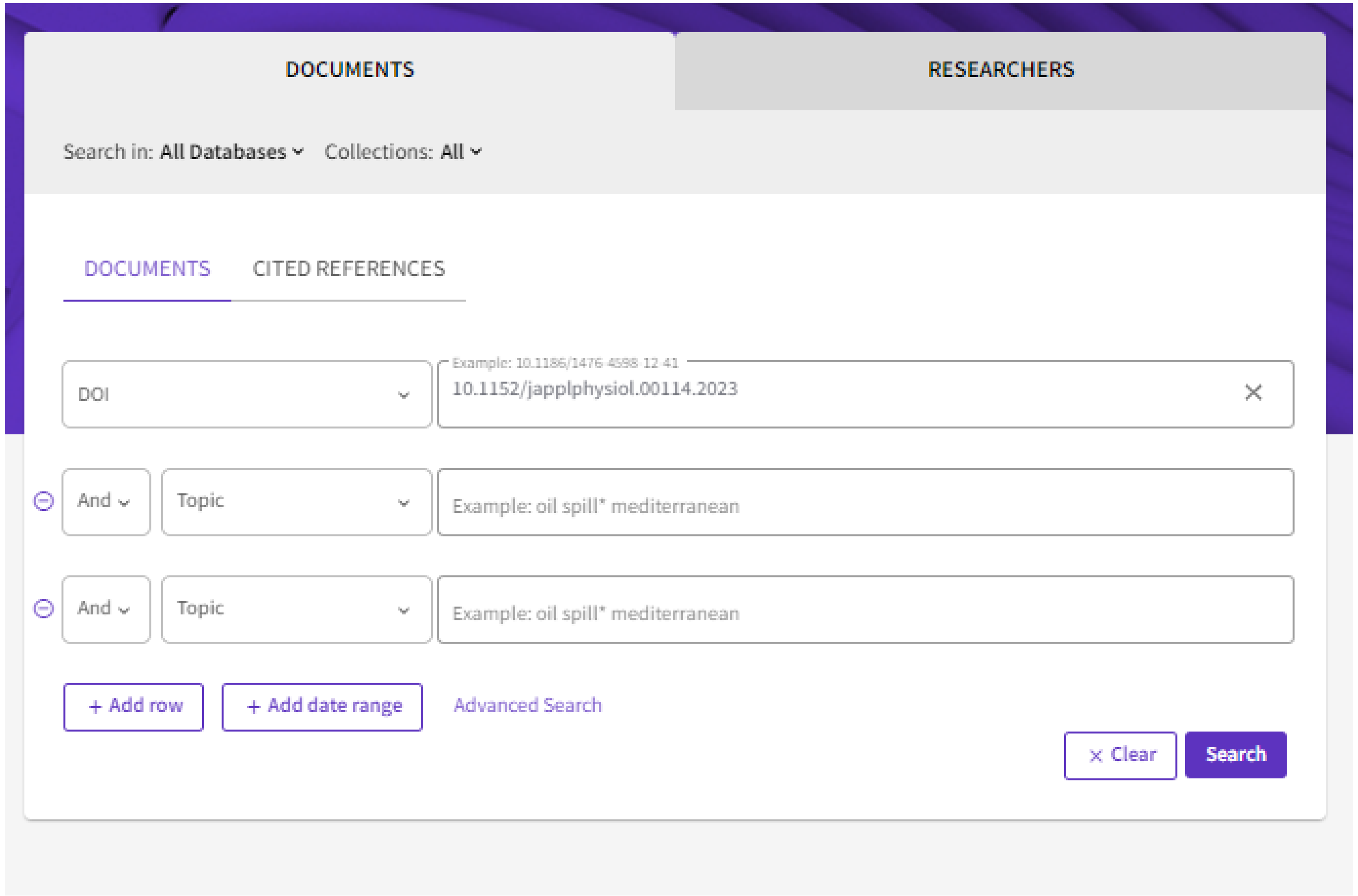

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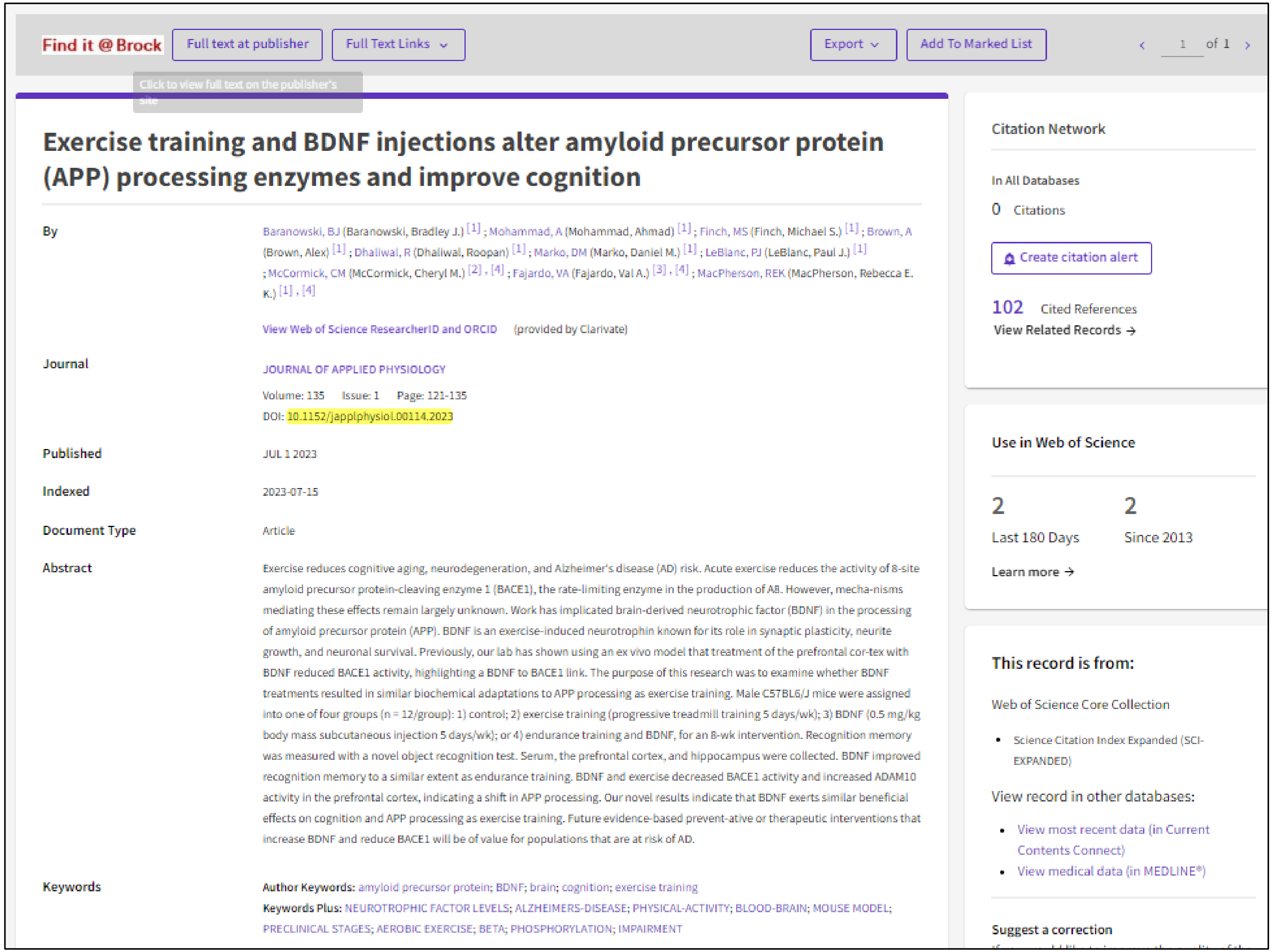

Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

By Baranowski, BJ (Baranowski, Bradley J.) [1] ; Mohammad, A (Mohammad, Ahmad) [1] ; Finch, MS (Finch, Michael S.) [1] ; Brown, A (Brown, Alex) [1] ; Dhaliwal, R (Dhaliwal, Roopan) [1] ; Marko, DM (Marko, Daniel M.) [1] ; LeBlanc, PJ (LeBlanc, Paul J.) [1] ; McCormick, CM (McCormick, Cheryl M.) [2] , [4] ; Fajardo, VA (Fajardo, Val A.) [3] , [4] ; MacPherson, REK (MacPherson, Rebecca E. K.) [1] , [4]

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Abstract Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of 8-site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of A8. However, mecha-nisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth, and neuronal survival. Previously, our lab has shown using an ex vivo model that treatment of the prefrontal cor-tex with BDNF reduced BACE1 activity, highlighting a BDNF to BACE1 link. The purpose of this research was to examine whether BDNF treatments resulted in similar biochemical adaptations to APP processing as exercise training. Male C57BL6/J mice were assigned into one of four groups (n = 12/group): 1) control; 2) exercise training (progressive treadmill training 5 days/wk); 3) BDNF (0.5 mg/kg body mass subcutaneous injection 5 days/wk); or 4) endurance training and BDNF, for an 8-wk intervention. Recognition memory was measured with a novel object recognition test. Serum, the prefrontal cortex, and hippocampus were collected. BDNF improved recognition memory to a similar extent as endurance training. BDNF and exercise decreased BACE1 activity and increased ADAM10 activity in the prefrontal cortex, indicating a shift in APP processing. Our novel results indicate that BDNF exerts similar beneficial effects on cognition and APP processing as exercise training. Future evidence-based prevent-ative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

Keywords **Author Keywords:** amyloid precursor protein; BDNF; brain; cognition; exercise training

Keywords Plus: NEUROTROPHIC FACTOR LEVELS; ALZHEIMERS-DISEASE; PHYSICAL-ACTIVITY; BLOOD-BRAIN; MOUSE MODEL; PRECLINICAL STAGES; AEROBIC EXERCISE; BETA; PHOSPHORYLATION; IMPAIRMENT

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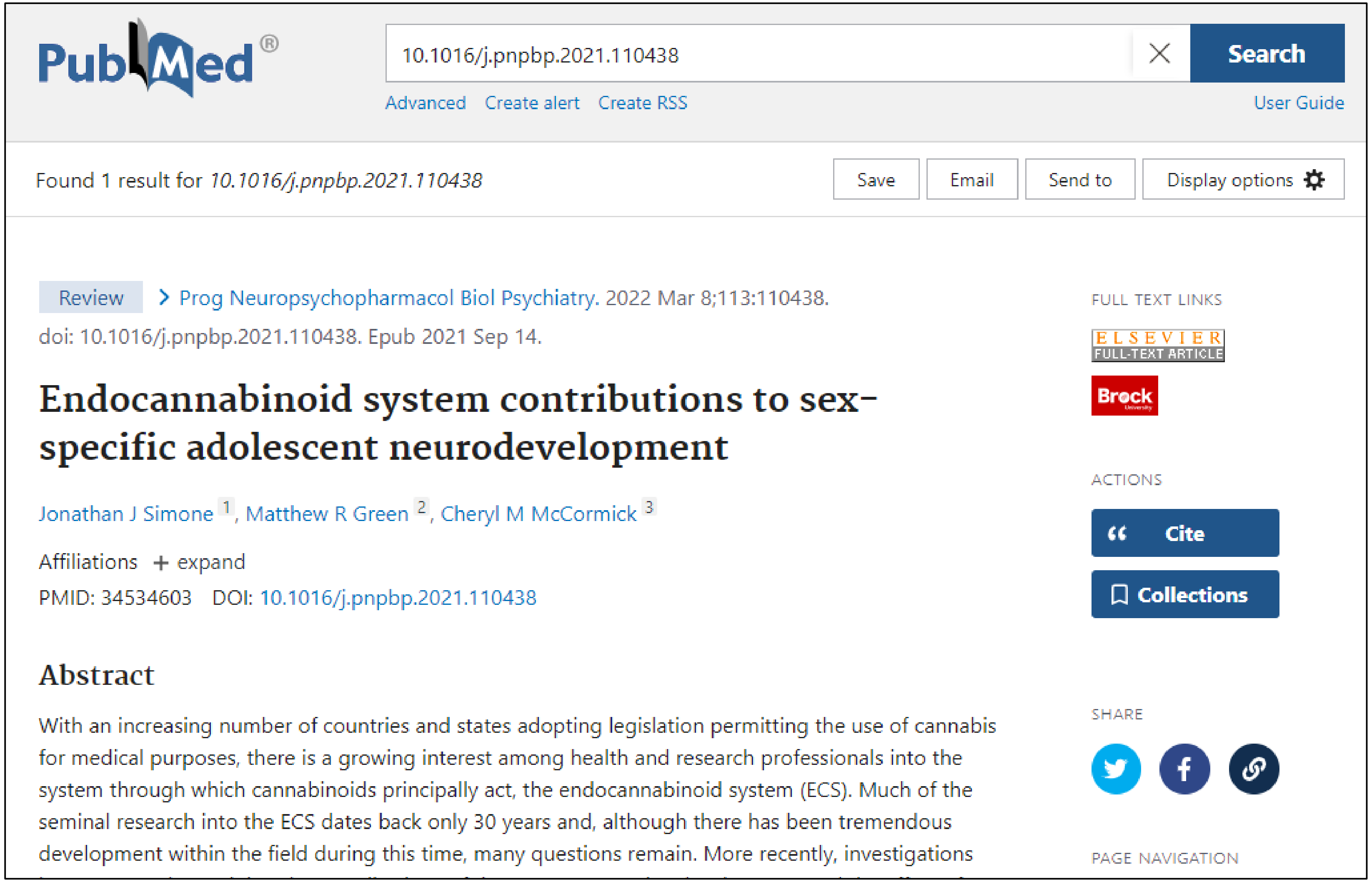

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Endocannabinoid system contributions to sex-specific adolescent neurodevelopment

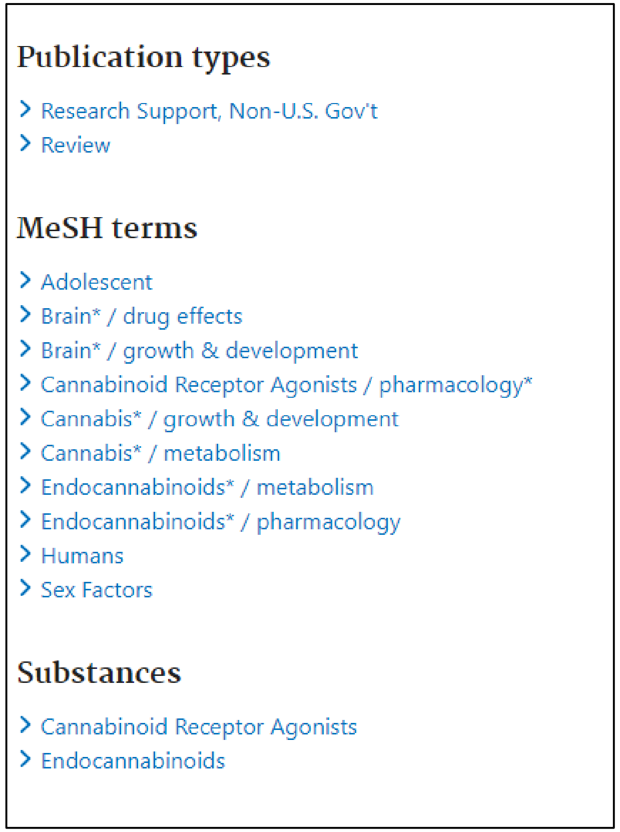
Jonathan J Simone [1], Matthew R Green [2], Cheryl M McCormick [3]

Affiliations + expand

PMID: 34534603 DOI: 10.1016/j.pnpbp.2021.110438

Abstract

With an increasing number of countries and states adopting legislation permitting the use of cannabis for medical purposes, there is a growing interest among health and research professionals into the system through which cannabinoids principally act, the endocannabinoid system (ECS). Much of the seminal research into the ECS dates back only 30 years and, although there has been tremendous development within the field during this time, many questions remain. More recently, investigations



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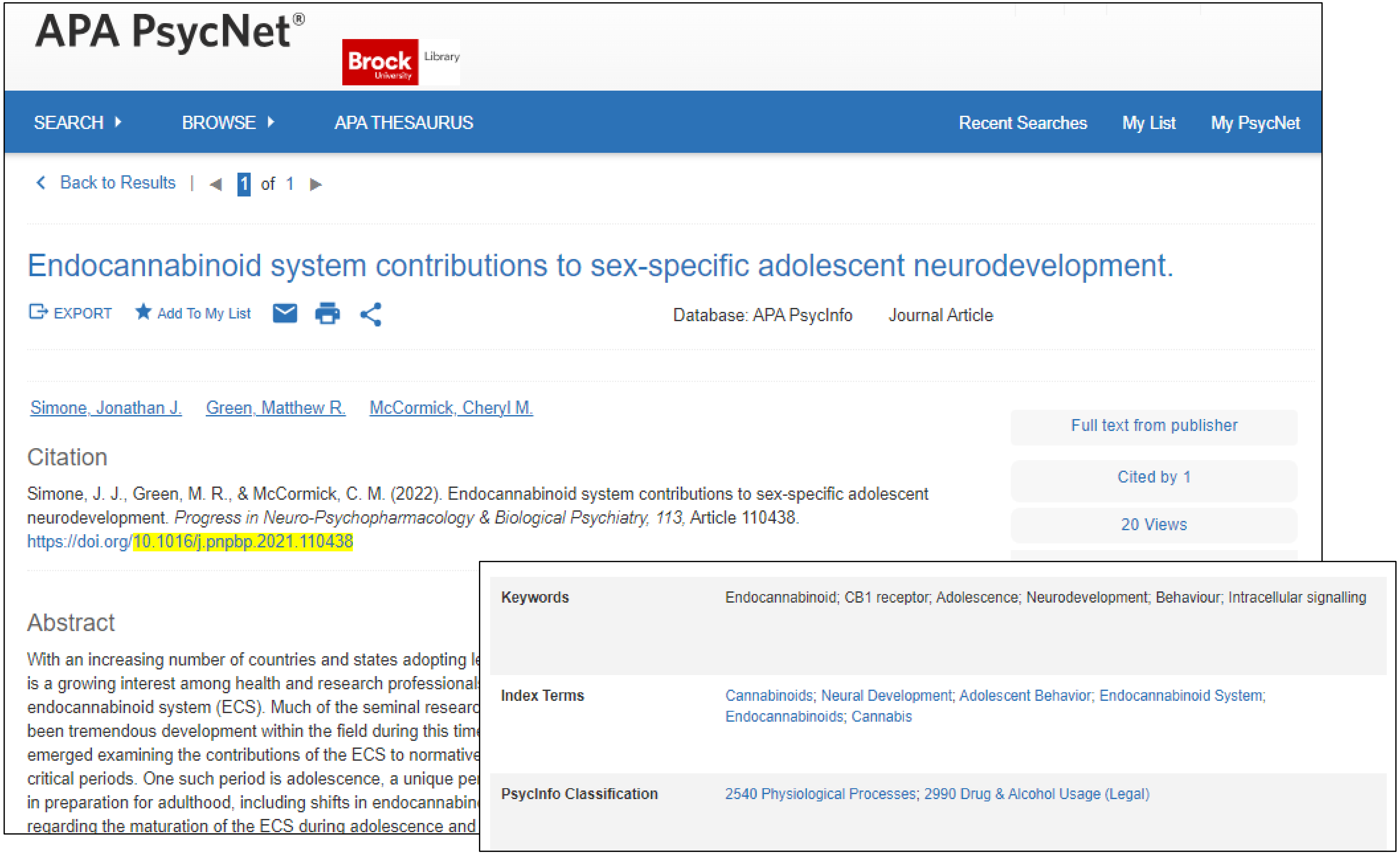
Simone, J. J., Green, M. R., & McCormick, C. M. (2022). Endocannabinoid system contributions to sex-specific adolescent neurodevelopment. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 113, 110438. https://doi.org/10.1016/j.pnpbp.2021.110438



Endocannabinoid system contributions to sex-specific adolescent neurodevelopment.

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Simone, Jonathan J. Green, Matthew R. McCormick, Cheryl M.

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Abstract

With an increasing number of countries and states adopting l
is a growing interest among health and research professional
endocannabinoid system (ECS). Much of the seminal researc
been tremendous development within the field during this tim
emerged examining the contributions of the ECS to normative
critical periods. One such period is adolescence, a unique pe
in preparation for adulthood, including shifts in endocannabin
regarding the maturation of the ECS during adolescence and

Keywords	Endocannabinoid; CB1 receptor; Adolescence; Neurodevelopment; Behaviour; Intracellular signalling
Index Terms	Cannabinoids; Neural Development; Adolescent Behavior; Endocannabinoid System; Endocannabinoids; Cannabis
PsycInfo Classification	2540 Physiological Processes; 2990 Drug & Alcohol Usage (Legal)

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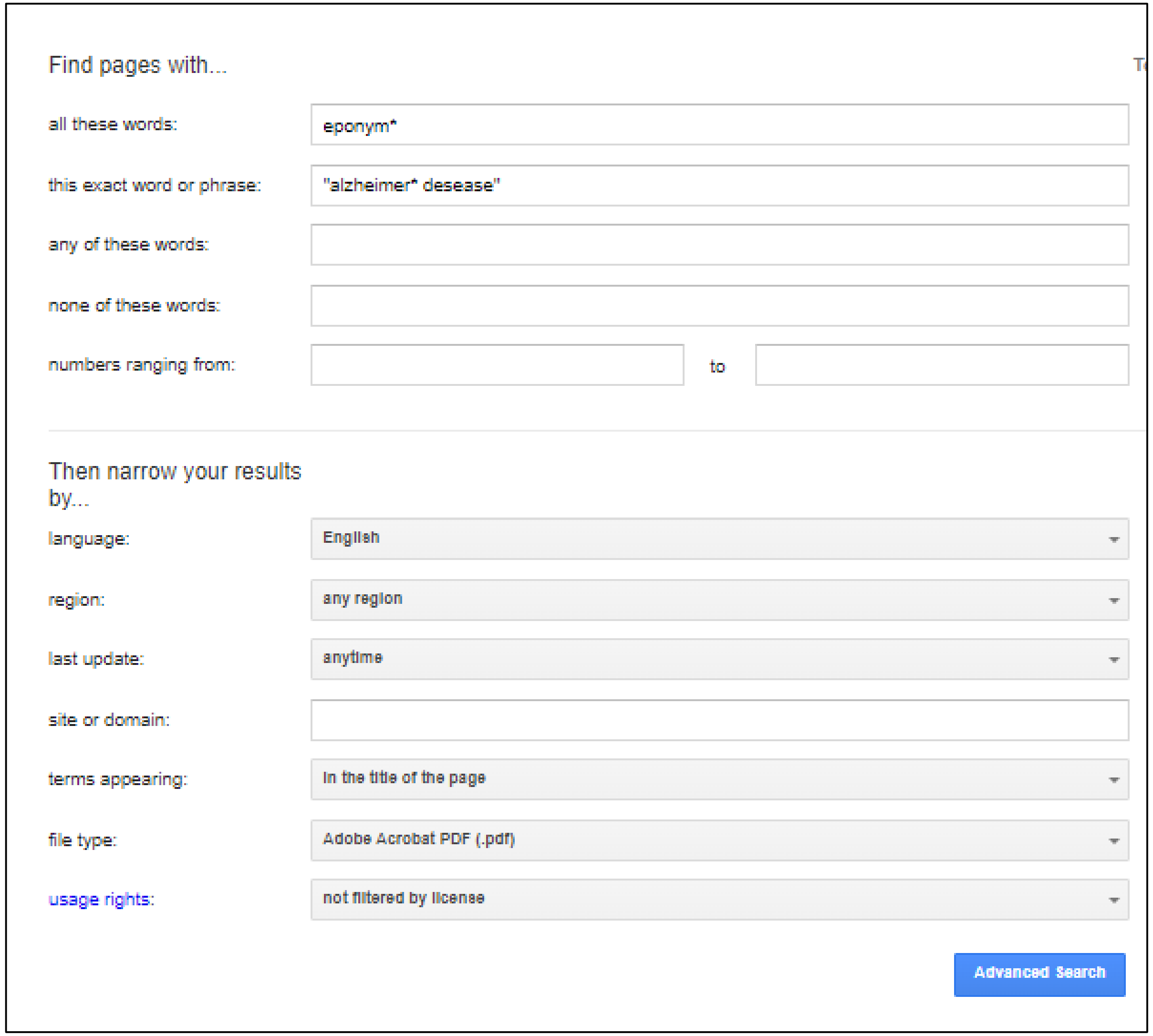

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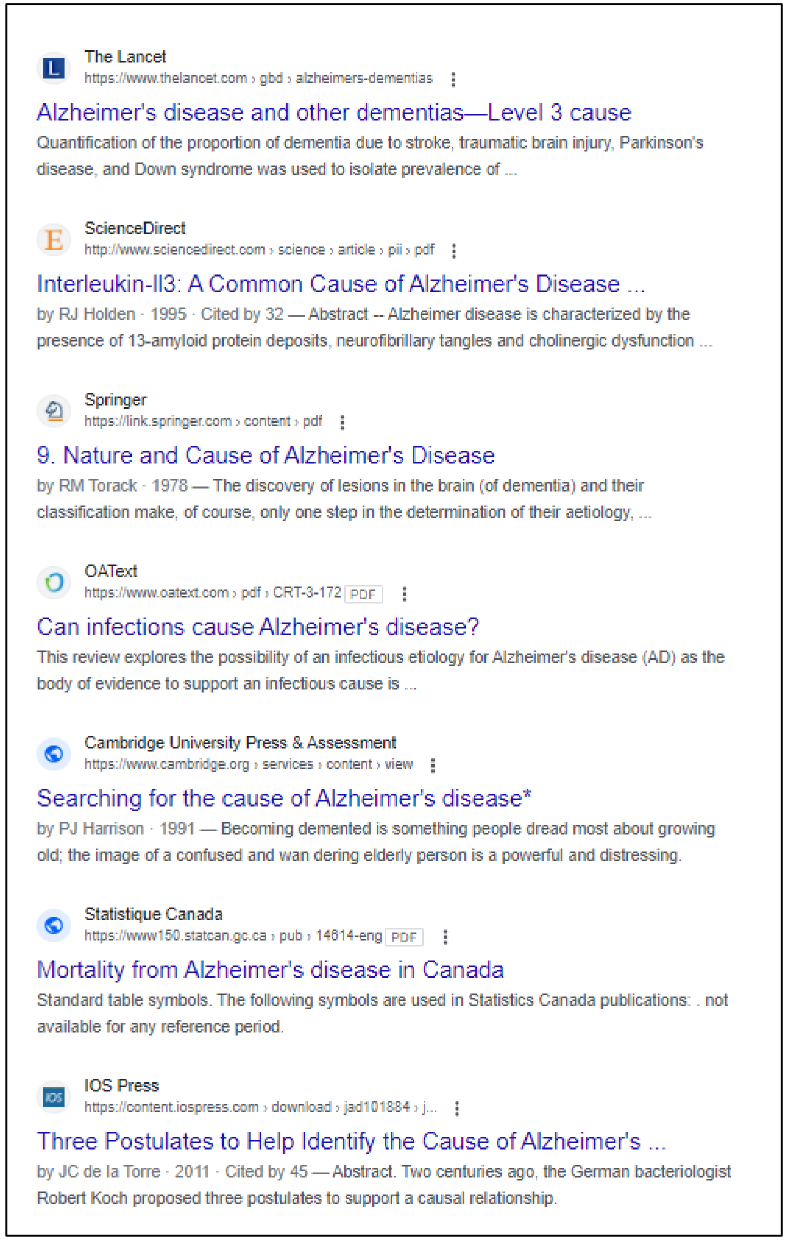

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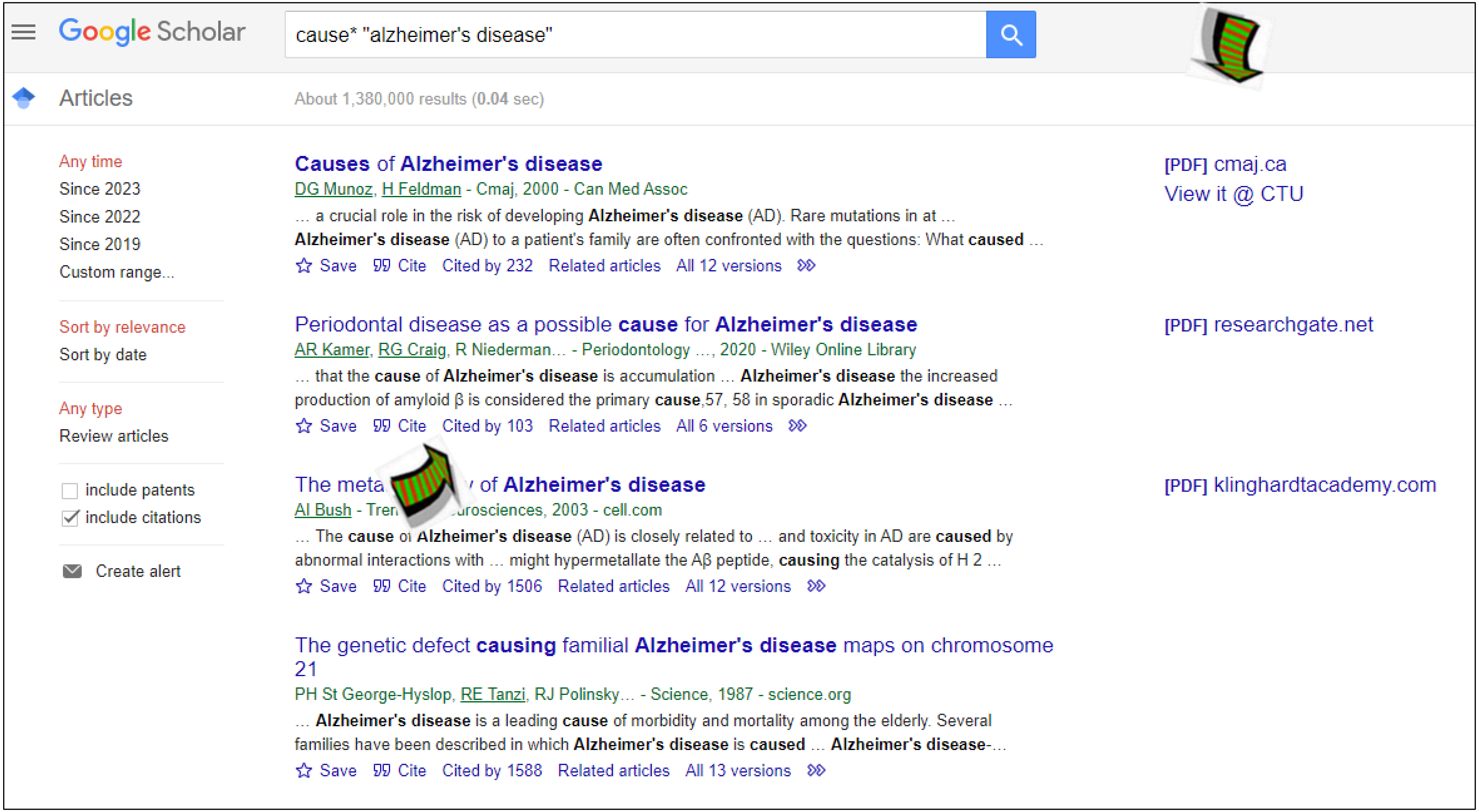

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- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers

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RESEARCH ARTICLE

Physical Activity and the Brain

Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

Bradley J. Baranowski,[1] Ahmad Mohammad,[1] Michael S. Finch,[1] Alex Brown,[1] Roopan Dhaliwal,[1] Daniel M. Marko,[1] Paul J. LeBlanc,[1] Cheryl M. McCormick,[2,4] Val A. Fajardo,[3,4] and Rebecca E. K. MacPherson[1,4]

[1]Department of Health Sciences, Brock University, St. Catharines, Ontario, Canada; [2]Department of Psychology, Brock University, St Catharines, Ontario, Canada; [3]Department of Kinesiology, Brock University, St Catharines, Ontario, Canada; and [4]Centre for Neuroscience, Brock University, St. Catharines, Ontario, Canada

Abstract

Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β-site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of Aβ. However, mechanisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth, and neuronal survival. Previously, our lab has shown using an ex vivo model that treatment of the prefrontal cortex with BDNF reduced BACE1 activity, highlighting a BDNF to BACE1 link. The purpose of this research was to examine whether BDNF treatments resulted in similar biochemical adaptations to APP processing as exercise training. Male C57BL6/J mice were assigned into one of four groups (n = 12/group): *1*) control; *2*) exercise training (progressive treadmill training 5 days/wk); *3*) BDNF (0.5 mg/kg body mass subcutaneous injection 5 days/wk); or *4*) endurance training and BDNF, for an 8-wk intervention. Recognition memory was measured with a novel object recognition test. Serum, the prefrontal cortex, and hippocampus were collected. BDNF improved recognition memory to a similar extent as endurance training. BDNF and exercise decreased BACE1 activity and increased ADAM10 activity in the prefrontal cortex, indicating a shift in APP processing. Our novel results indicate that BDNF exerts similar beneficial effects on cognition and APP processing as exercise training. Future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

NEW & NOTEWORTHY Our study presents the novel findings that chronic peripheral BDNF injections result in regulation of APP processing enzymes and improved cognition to a similar extent as exercise training. These findings highlight the potential efficacy of using BDNF as a therapeutic intervention in the prevention of neurodegenerative diseases (i.e., Alzheimer's disease). Furthermore, future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

amyloid precursor protein; BDNF; brain; cognition; exercise training

INTRODUCTION

The overproduction and accumulation of amyloid beta (Aβ) peptides that can result in plaque formation is a key feature of neurodegenerative diseases, such as Alzheimer's disease (AD). Aβ peptides have been shown to lead to neuronal loss, cognitive impairments, neuroinflammation, synaptic dysfunction, and other neurotoxic events in the brain (1). Alternatively, a lifelong underproduction of Aβ, as observed in individuals with the Icelandic mutation in amyloid precursor protein (APP), protects individuals from cognitive decline with aging (2). Increased production of these peptides can be observed decades before clinical diagnosis (3–6), highlighting the importance of early, preventative measures to reduce the Aβ burden as one ages. Aβ originates from a large transmembrane protein, known as the APP (7, 8). The fate of APP processing is determined by one of two competing pathways: the amyloidogenic and nonamyloidogenic pathways (9). The amyloidogenic pathway is initiated when the rate-limiting enzyme, β-site amyloid precursor protein cleaving enzyme 1 (BACE1), cleaves APP resulting in the release of a soluble ectodomain, soluble APPβ (sAPPβ) fragment, and leaving behind a membrane-bound C-terminal fragment (CTF) 89/99 (10, 11). ϒ-Secretase then cleaves the CTF89/99 fragment and releases

Correspondence: R. E. K. MacPherson (rmacpherson@brocku.ca).
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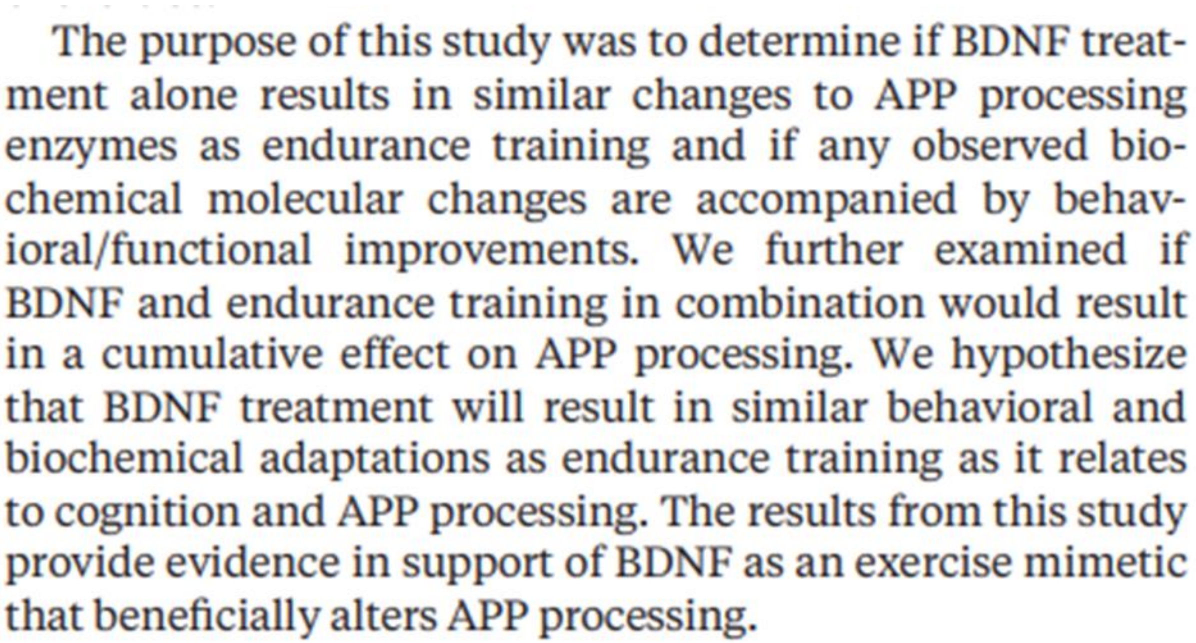
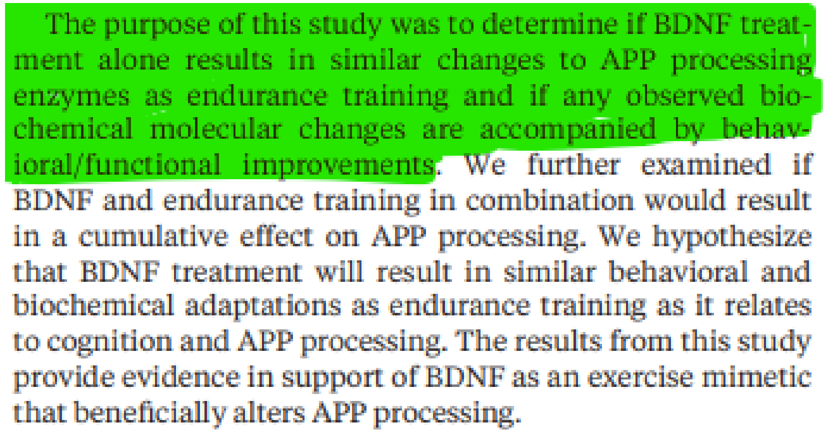


The purpose of this study was to determine if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements. We further examined if BDNF and endurance training in combination would result in a cumulative effect on APP processing. We hypothesize that BDNF treatment will result in similar behavioral and biochemical adaptations as endurance training as it relates to cognition and APP processing. The results from this study provide evidence in support of BDNF as an exercise mimetic that beneficially alters APP processing.

My investigation attempts to find literature that helps to explain "…if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements" (Baranowski et al, 2023, 122).

Baranowski, B. J., Mohammad, A., Finch, M. S., Brown, A., Dhaliwal, R., Marko, D. M., LeBlanc, P. J., McCormick, C. M., Fajardo, V. A., & MacPherson, R. E. K. (2023). Exercise training and BDNF injections alter amyloid precursor protein (App) processing enzymes and improve cognition. *Journal of Applied Physiology, 135*(1), 121–135. https://doi.org/10.1152/japplphysiol.00114.2023



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2. APP processing enzymes
3. Biochemical molecular changes
4. Behavioral/functional improvements

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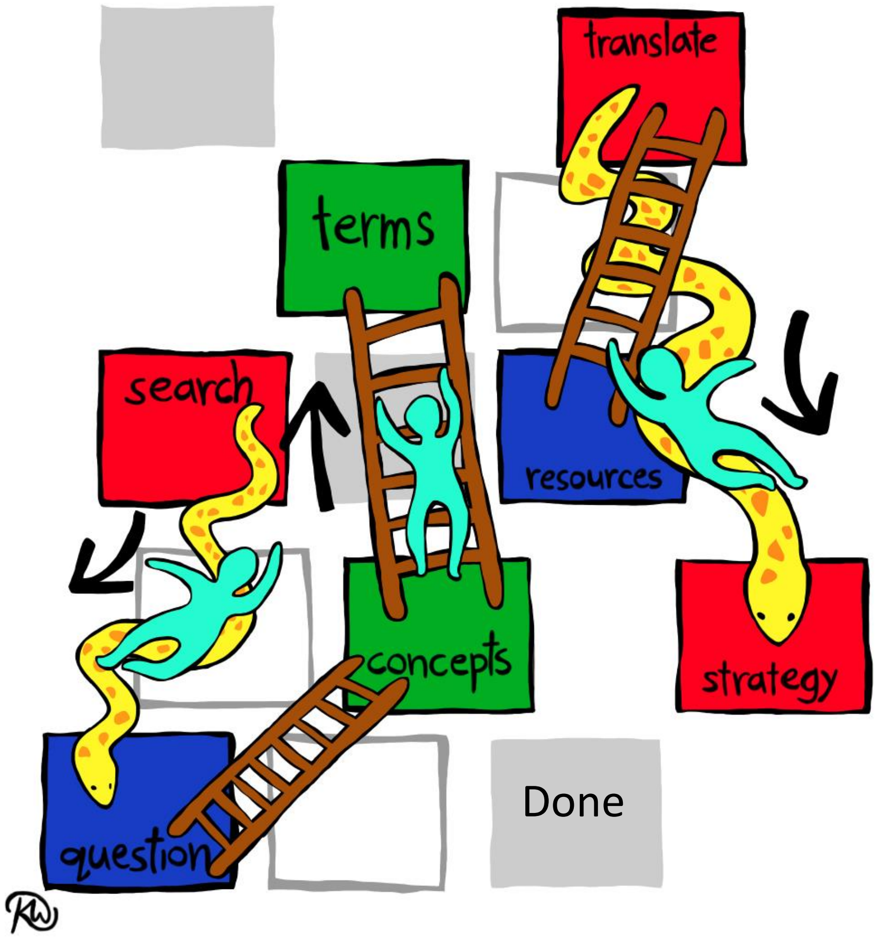

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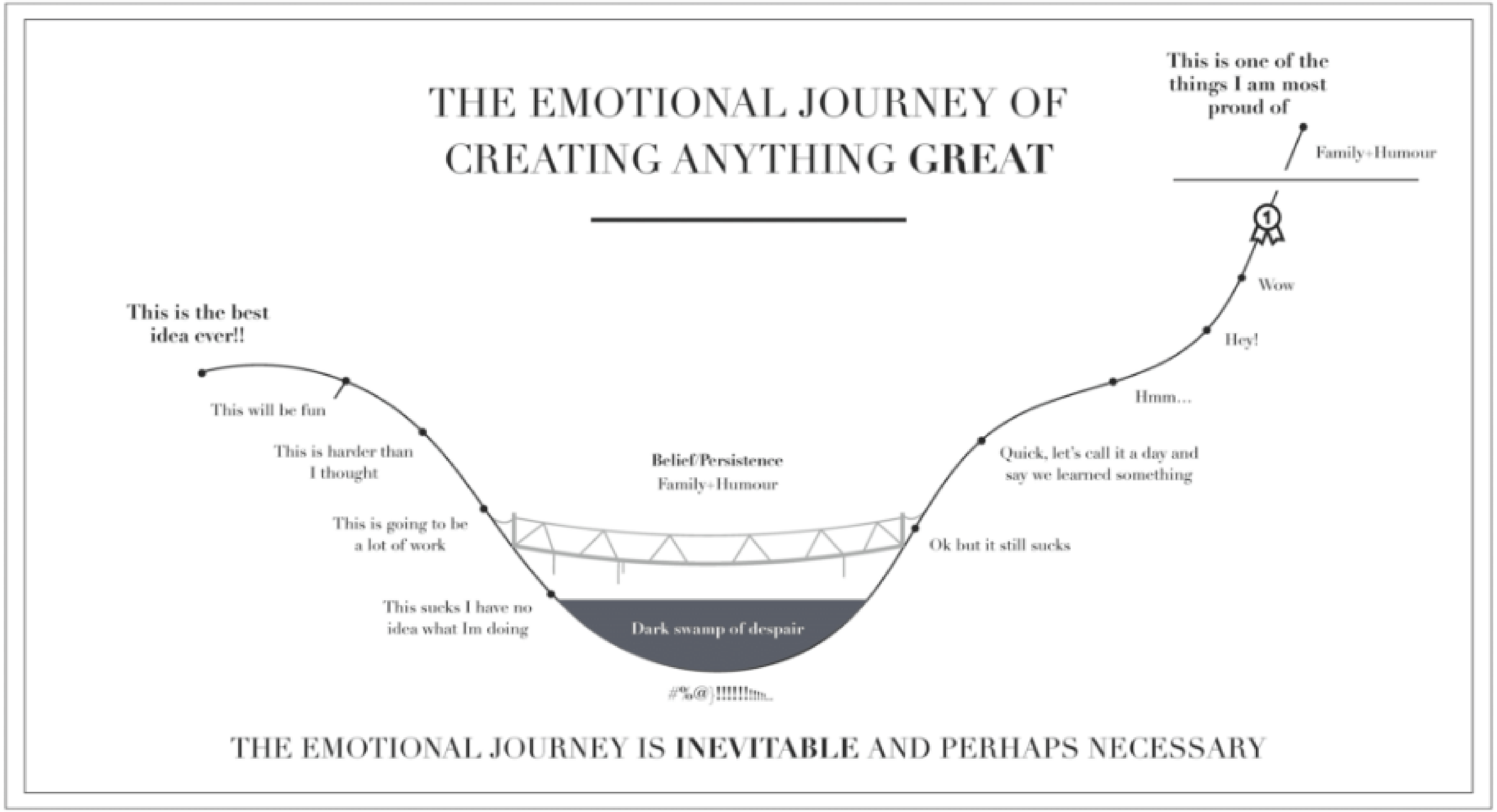
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"…if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements"

1. BDNF treatment(s)
2. APP processing enzymes
3. Biochemical molecular changes
4. Behavioral/functional improvements

"brain-derived neurotrophic factor" or BDNF* or neurotrophin*

"amyloid precursor protein*" or APP* or "processing enzyme*"

Neurodegenerative or Alzheimer* or "neuronal" or "cognitive impairment*" or neuroinflammation or "synaptic dysfunction*" or neurotoxic*

brain or nerve* or cognitive*

behav* or function*

5. Scholarly articles
6. 1990 to the present
7. English Language
8. Neurology/Biology/Medicine…
9. Databases…
10. Search strategies…
11. Zoterobib
12. Zotero

2000+
English language
Neurology and interdisciplinary databases
WofS Complete, PsycINFO, GS

"…if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements"

1. BDNF treatment(s)
2. APP processing enzymes
3. Biochemical molecular changes
4. Behavioral/functional improvements

"brain-derived neurotrophic factor" or BDNF* or neurotrophin*

"amyloid precursor protein*" or APP* or "processing enzyme*"

Neurodegenerative or Alzheimer* or "neuronal" or "cognitive impairment*" or neuroinflammation or "synaptic dysfunction*" or neurotoxic*

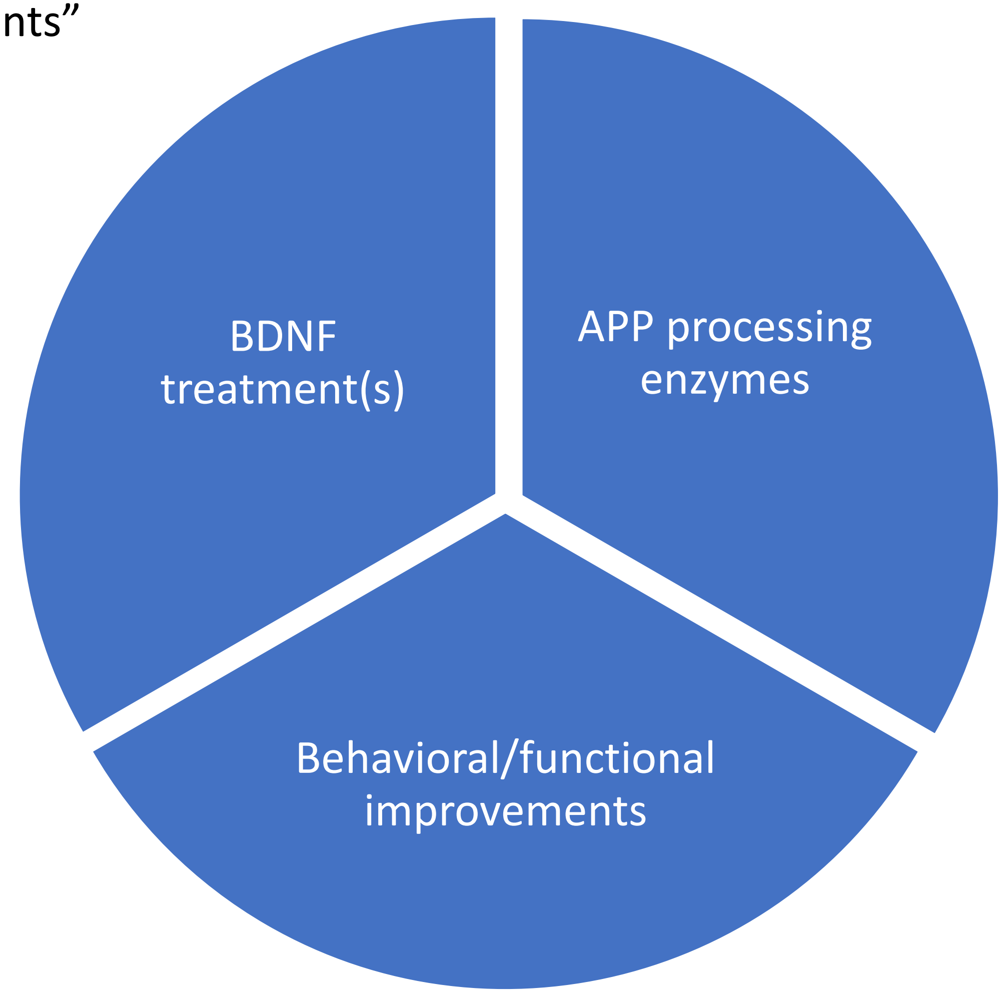
brain or nerve* or cognitive*

behav* or function*

5. Scholarly articles
6. 1990 to the present
7. English Language
8. Neurology/Biology/Medicine…
9. Databases…
10. Search strategies…
11. Zoterobib
12. Zotero

2000+
English language
Neurology and interdisciplinary databases
WofS Complete, PsycINFO, GS

"…if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements"



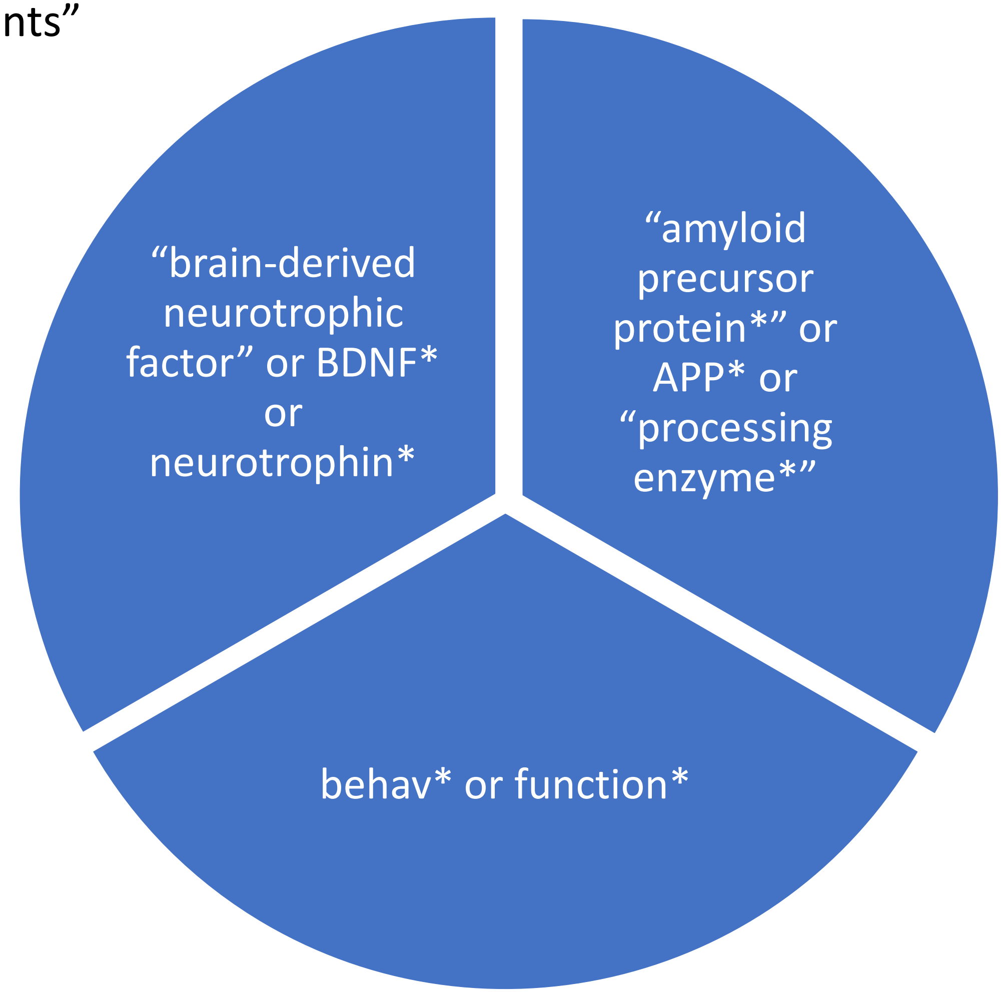
BDNF treatment(s)

APP processing enzymes

Behavioral/functional improvements

2000+
English language
WofS Complete

"…if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements"

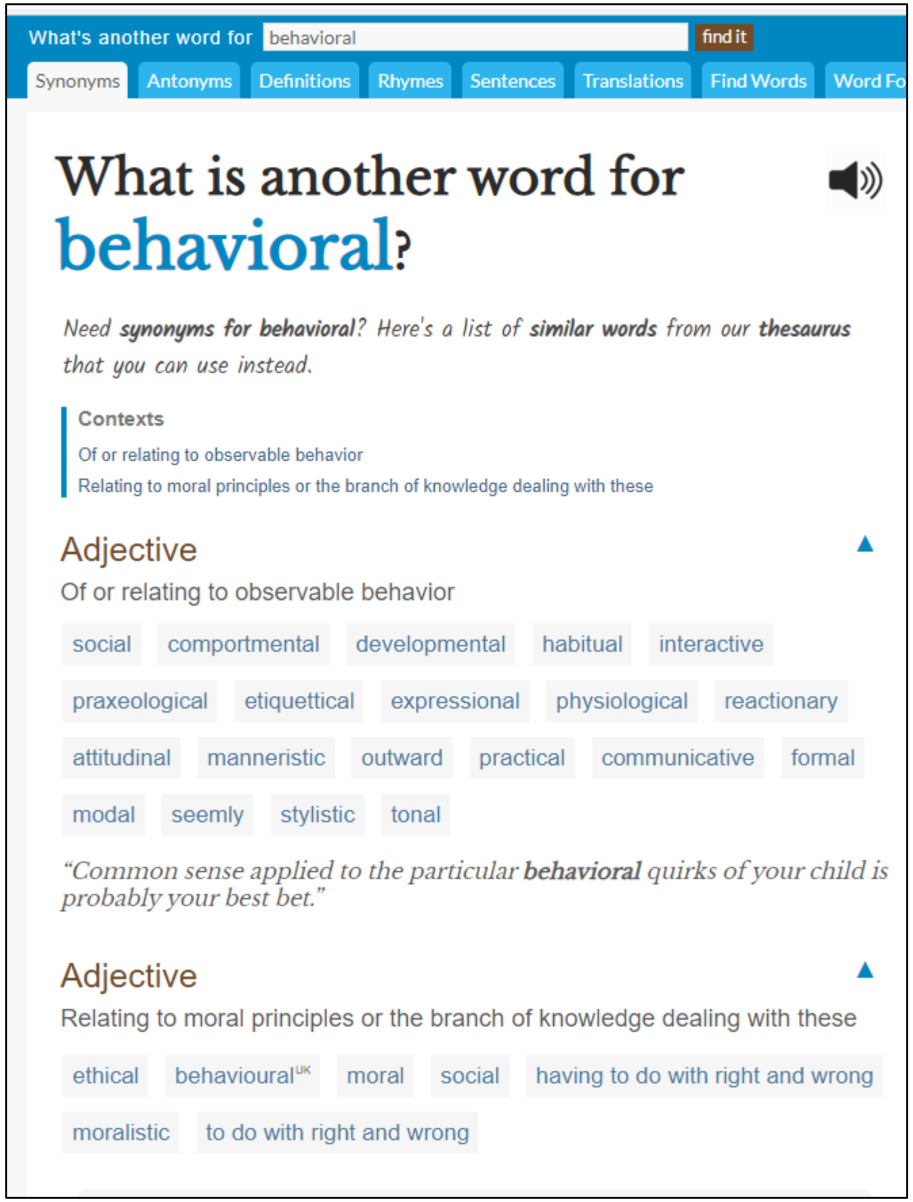


"brain-derived neurotrophic factor" or BDNF* or neurotrophin*

"amyloid precursor protein*" or APP* or "processing enzyme*"

behav* or function*

2000+
English language
WofS Complete



What is another word for behavioral?

*Need **synonyms for behavioral**? Here's a list of **similar words** from our **thesaurus** that you can use instead.*

Contexts

Of or relating to observable behavior

Relating to moral principles or the branch of knowledge dealing with these

Adjective

Of or relating to observable behavior

social · comportmental · developmental · habitual · interactive · praxeological · etiquettical · expressional · physiological · reactionary · attitudinal · manneristic · outward · practical · communicative · formal · modal · seemly · stylistic · tonal

*"Common sense applied to the particular **behavioral** quirks of your child is probably your best bet."*

Adjective

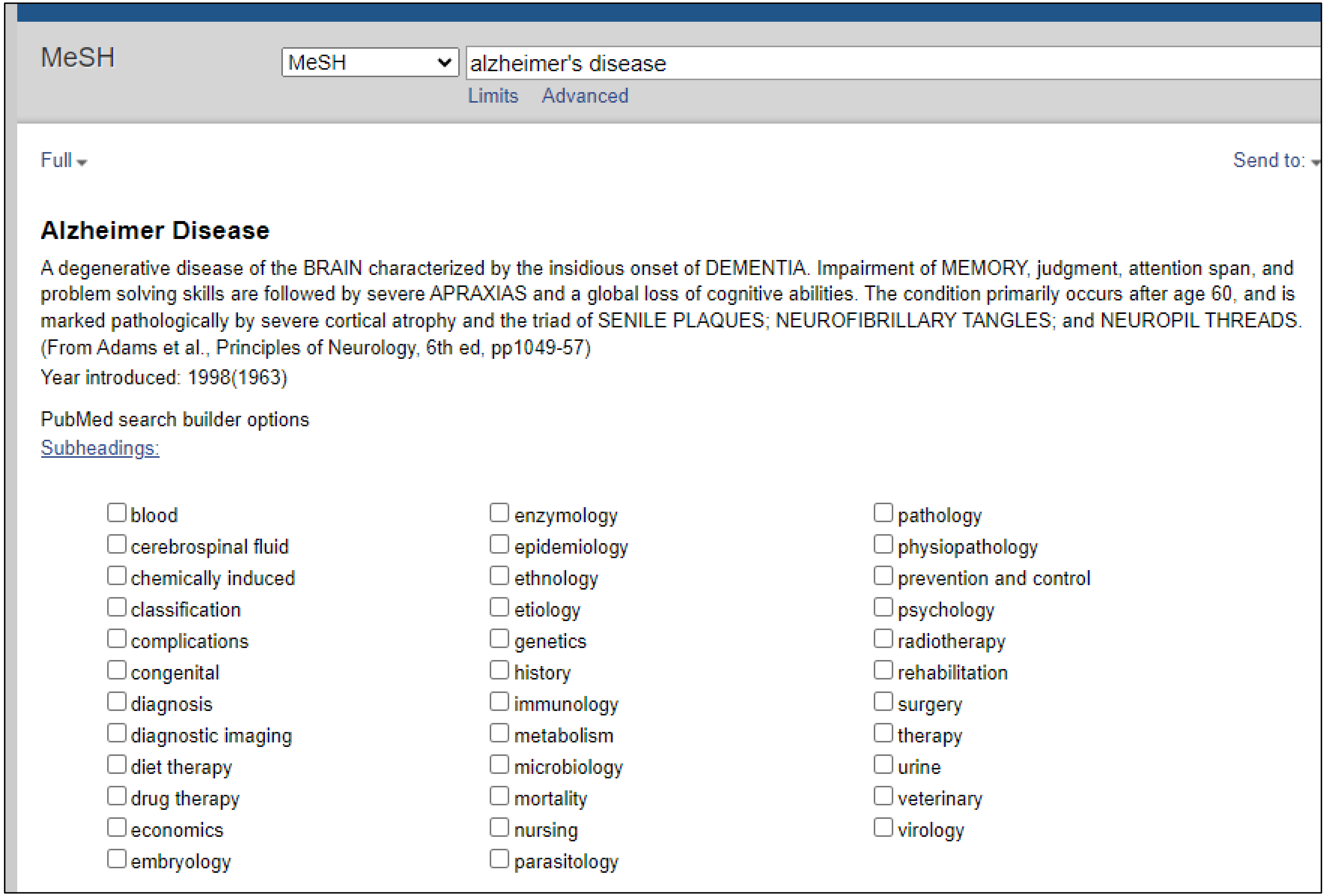
Relating to moral principles or the branch of knowledge dealing with these

ethical · behavioural^{UK} · moral · social · having to do with right and wrong · moralistic · to do with right and wrong

Word hIPPO

https://www.wordhippo.com/

https://www.ncbi.nlm.nih.gov/mesh



alzheimer's disease

Alzheimer Disease

A degenerative disease of the BRAIN characterized by the insidious onset of DEMENTIA. Impairment of MEMORY, judgment, attention span, and problem solving skills are followed by severe APRAXIAS and a global loss of cognitive abilities. The condition primarily occurs after age 60, and is marked pathologically by severe cortical atrophy and the triad of SENILE PLAQUES; NEUROFIBRILLARY TANGLES; and NEUROPIL THREADS. (From Adams et al., Principles of Neurology, 6th ed, pp1049-57)

Year introduced: 1998(1963)

PubMed search builder options

Subheadings:

- ☐ blood
- ☐ cerebrospinal fluid
- ☐ chemically induced
- ☐ classification
- ☐ complications
- ☐ congenital
- ☐ diagnosis
- ☐ diagnostic imaging
- ☐ diet therapy
- ☐ drug therapy
- ☐ economics
- ☐ embryology
- ☐ enzymology
- ☐ epidemiology
- ☐ ethnology
- ☐ etiology
- ☐ genetics
- ☐ history
- ☐ immunology
- ☐ metabolism
- ☐ microbiology
- ☐ mortality
- ☐ nursing
- ☐ parasitology
- ☐ pathology
- ☐ physiopathology
- ☐ prevention and control
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- ☐ surgery
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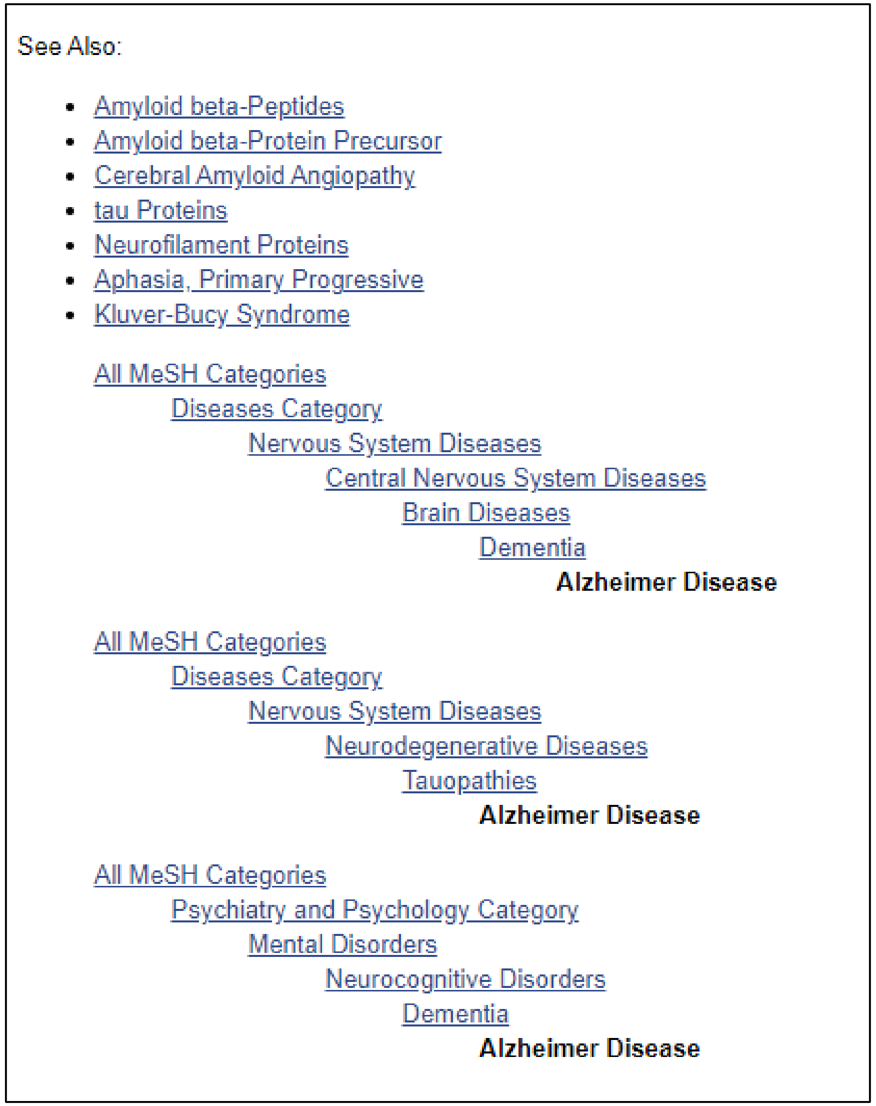
- ☐ urine
- ☐ veterinary
- ☐ virology



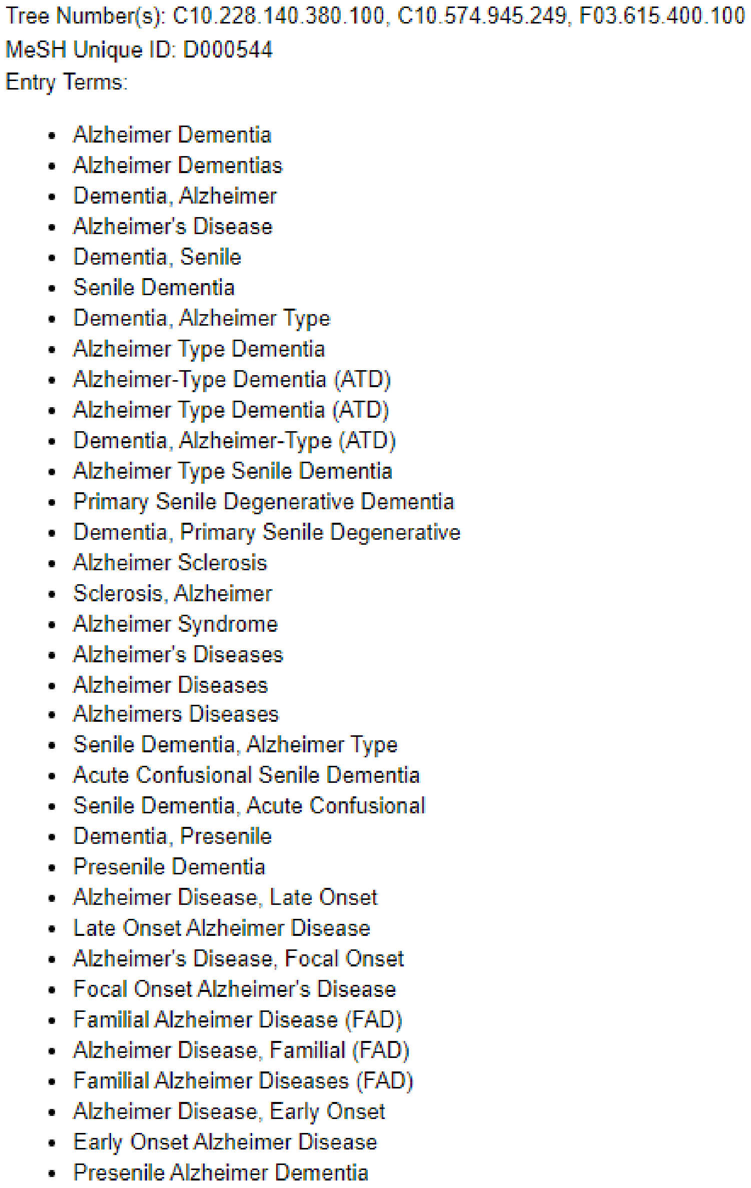
See Also:

- Amyloid beta-Peptides
- Amyloid beta-Protein Precursor
- Cerebral Amyloid Angiopathy
- tau Proteins
- Neurofilament Proteins
- Aphasia, Primary Progressive
- Kluver-Bucy Syndrome

All MeSH Categories
Diseases Category
Nervous System Diseases
Central Nervous System Diseases
Brain Diseases
Dementia
Alzheimer Disease

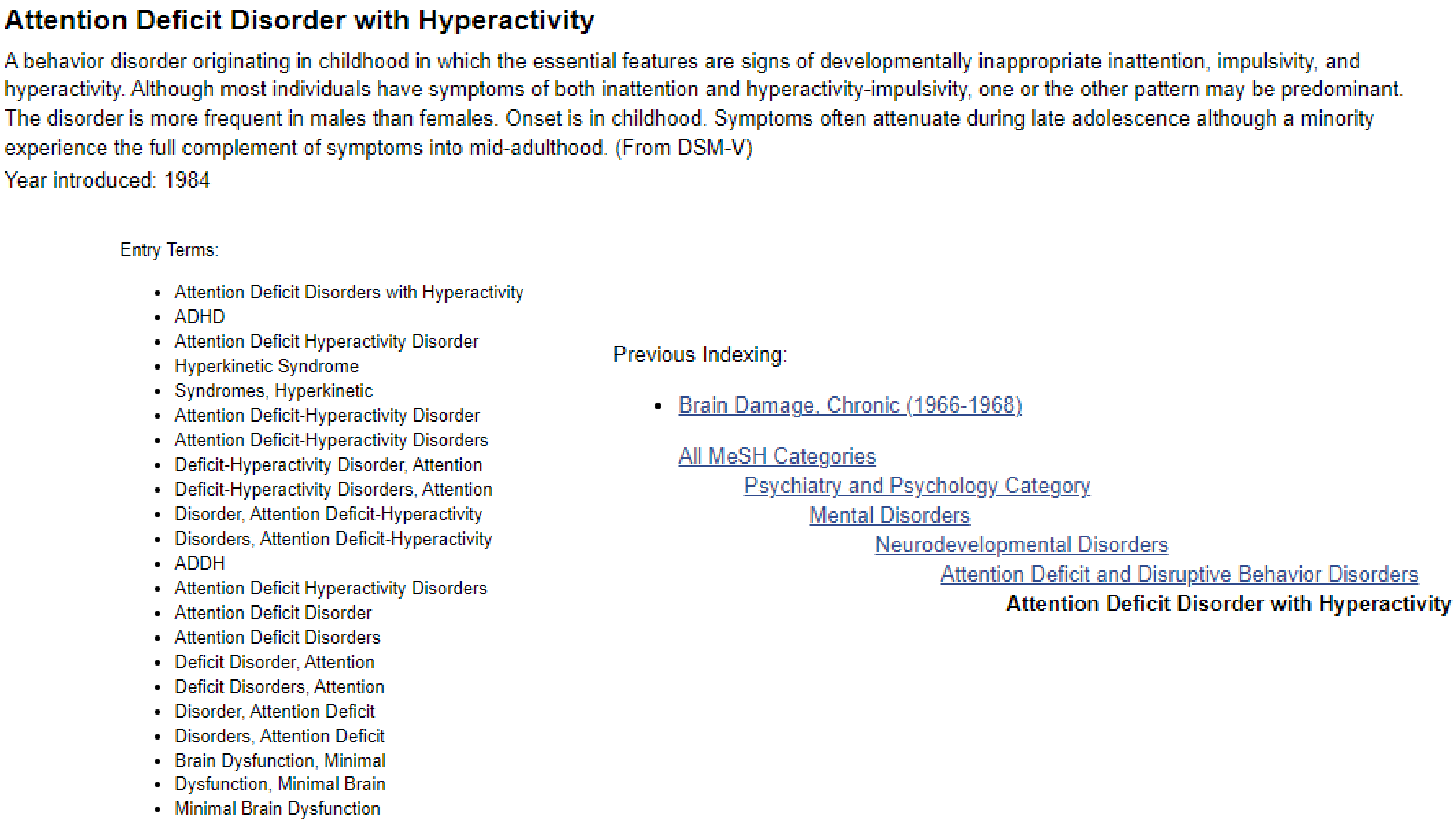
All MeSH Categories
Diseases Category
Nervous System Diseases
Neurodegenerative Diseases
Tauopathies
Alzheimer Disease

All MeSH Categories
Psychiatry and Psychology Category
Mental Disorders
Neurocognitive Disorders
Dementia
Alzheimer Disease



Tree Number(s): C10.228.140.380.100, C10.574.945.249, F03.615.400.100
MeSH Unique ID: D000544
Entry Terms:

- Alzheimer Dementia
- Alzheimer Dementias
- Dementia, Alzheimer
- Alzheimer's Disease
- Dementia, Senile
- Senile Dementia
- Dementia, Alzheimer Type
- Alzheimer Type Dementia
- Alzheimer-Type Dementia (ATD)
- Alzheimer Type Dementia (ATD)
- Dementia, Alzheimer-Type (ATD)
- Alzheimer Type Senile Dementia
- Primary Senile Degenerative Dementia
- Dementia, Primary Senile Degenerative
- Alzheimer Sclerosis
- Sclerosis, Alzheimer
- Alzheimer Syndrome
- Alzheimer's Diseases
- Alzheimer Diseases
- Alzheimers Diseases
- Senile Dementia, Alzheimer Type
- Acute Confusional Senile Dementia
- Senile Dementia, Acute Confusional
- Dementia, Presenile
- Presenile Dementia
- Alzheimer Disease, Late Onset
- Late Onset Alzheimer Disease
- Alzheimer's Disease, Focal Onset
- Focal Onset Alzheimer's Disease
- Familial Alzheimer Disease (FAD)
- Alzheimer Disease, Familial (FAD)
- Familial Alzheimer Diseases (FAD)
- Alzheimer Disease, Early Onset
- Early Onset Alzheimer Disease
- Presenile Alzheimer Dementia



Attention Deficit Disorder with Hyperactivity

A behavior disorder originating in childhood in which the essential features are signs of developmentally inappropriate inattention, impulsivity, and hyperactivity. Although most individuals have symptoms of both inattention and hyperactivity-impulsivity, one or the other pattern may be predominant. The disorder is more frequent in males than females. Onset is in childhood. Symptoms often attenuate during late adolescence although a minority experience the full complement of symptoms into mid-adulthood. (From DSM-V)

Year introduced: 1984

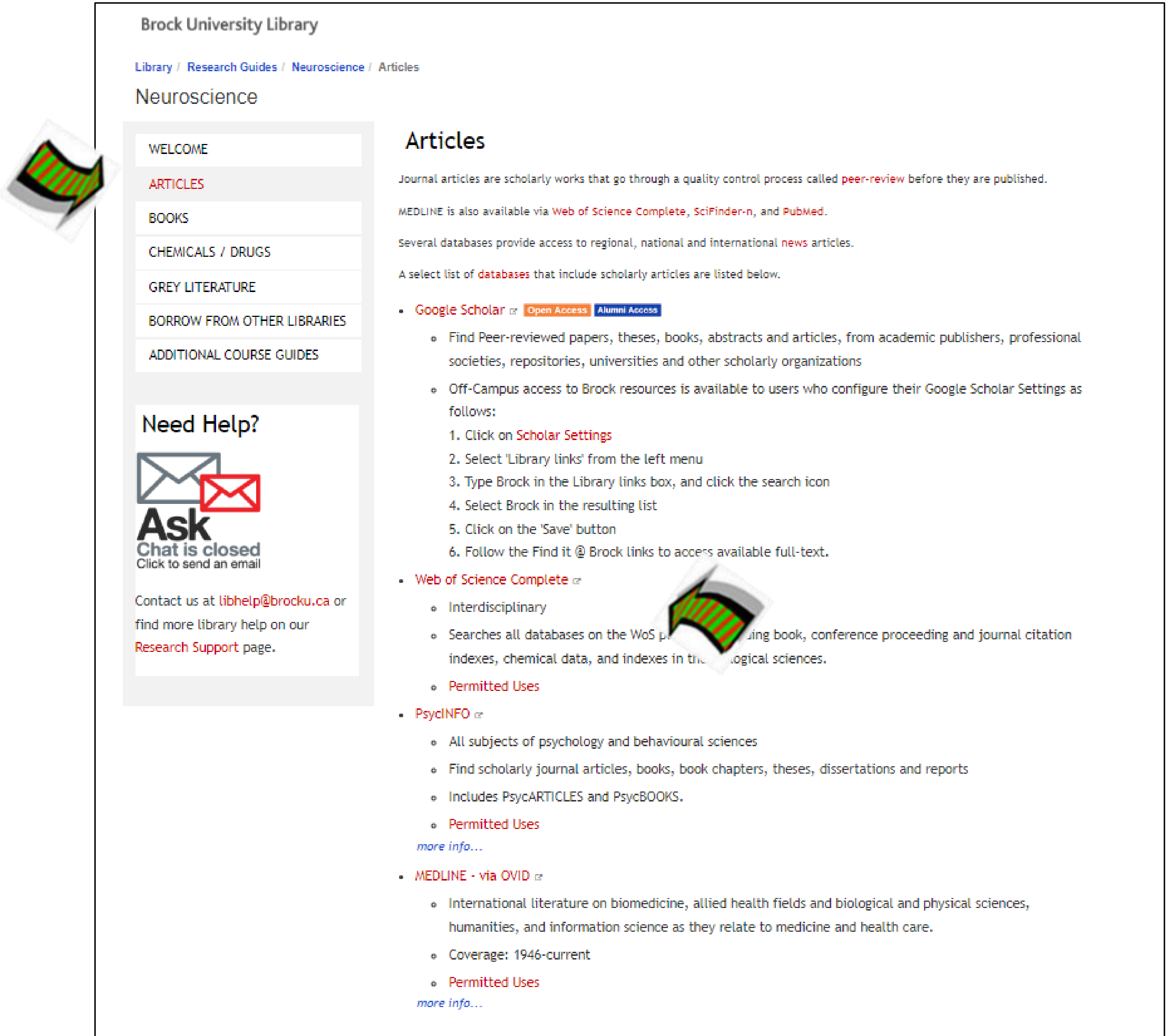
Entry Terms:

- Attention Deficit Disorders with Hyperactivity
- ADHD
- Attention Deficit Hyperactivity Disorder
- Hyperkinetic Syndrome
- Syndromes, Hyperkinetic
- Attention Deficit-Hyperactivity Disorder
- Attention Deficit-Hyperactivity Disorders
- Deficit-Hyperactivity Disorder, Attention
- Deficit-Hyperactivity Disorders, Attention
- Disorder, Attention Deficit-Hyperactivity
- Disorders, Attention Deficit-Hyperactivity
- ADDH
- Attention Deficit Hyperactivity Disorders
- Attention Deficit Disorder
- Attention Deficit Disorders
- Deficit Disorder, Attention
- Deficit Disorders, Attention
- Disorder, Attention Deficit
- Disorders, Attention Deficit
- Brain Dysfunction, Minimal
- Dysfunction, Minimal Brain
- Minimal Brain Dysfunction

Previous Indexing:

- Brain Damage, Chronic (1966-1968)

All MeSH Categories
- Psychiatry and Psychology Category
  - Mental Disorders
    - Neurodevelopmental Disorders
      - Attention Deficit and Disruptive Behavior Disorders
        - **Attention Deficit Disorder with Hyperactivity**



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Neuroscience

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Articles

Journal articles are scholarly works that go through a quality control process called peer-review before they are published.

MEDLINE is also available via Web of Science Complete, SciFinder-n, and PubMed.

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A select list of databases that include scholarly articles are listed below.

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    3. Type Brock in the Library links box, and click the search icon
    4. Select Brock in the resulting list
    5. Click on the 'Save' button
    6. Follow the Find it @ Brock links to access available full-text.
- Web of Science Complete
  - Interdisciplinary
  - Searches all databases on the WoS p ing book, conference proceeding and journal citation indexes, chemical data, and indexes in th logical sciences.
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- PsycINFO
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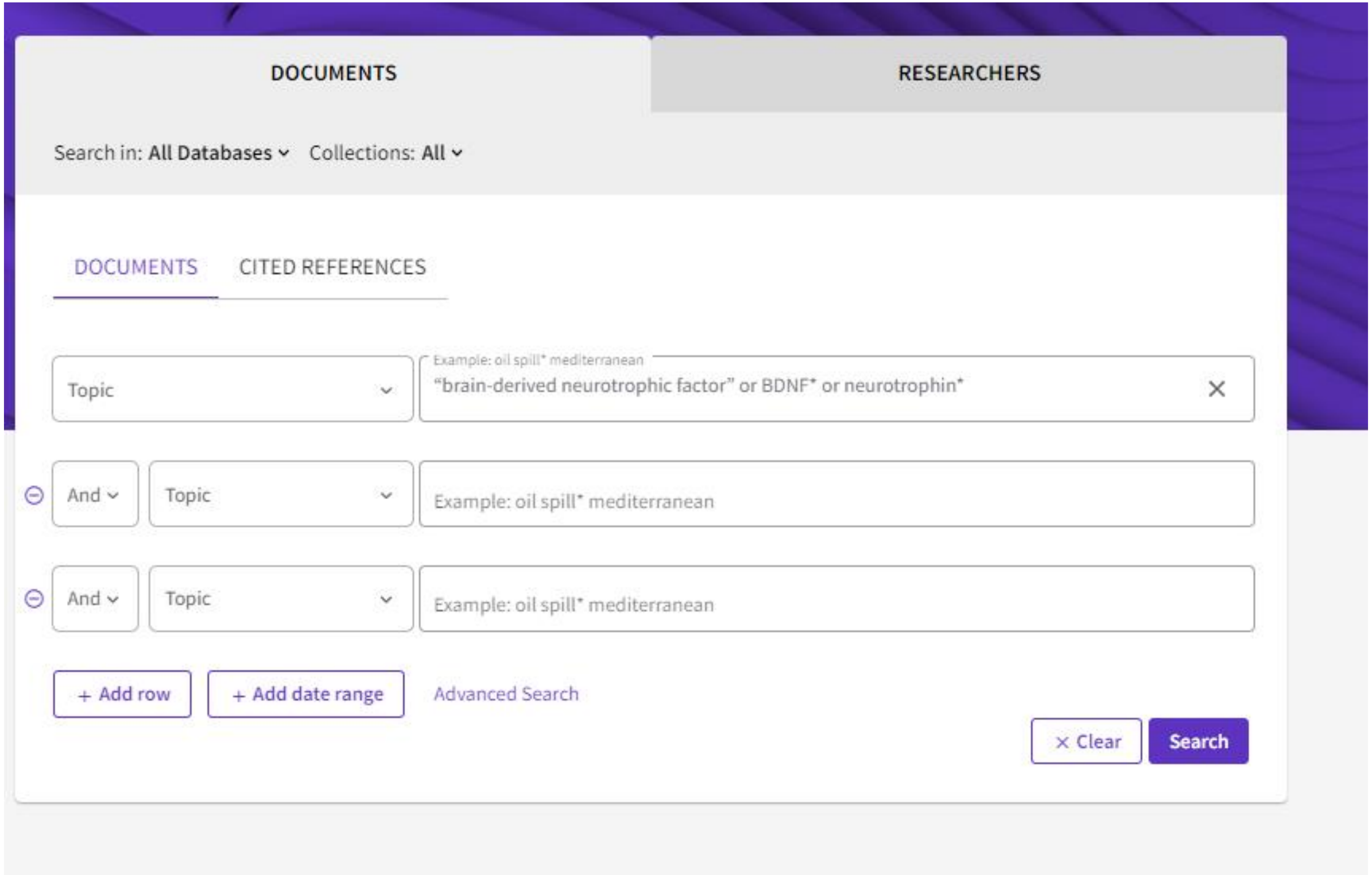
  - Find scholarly journal articles, books, book chapters, theses, dissertations and reports
  - Includes PsycARTICLES and PsycBOOKS.
  - Permitted Uses

  more info...
- MEDLINE - via OVID
  - International literature on biomedicine, allied health fields and biological and physical sciences, humanities, and information science as they relate to medicine and health care.
  - Coverage: 1946-current
  - Permitted Uses

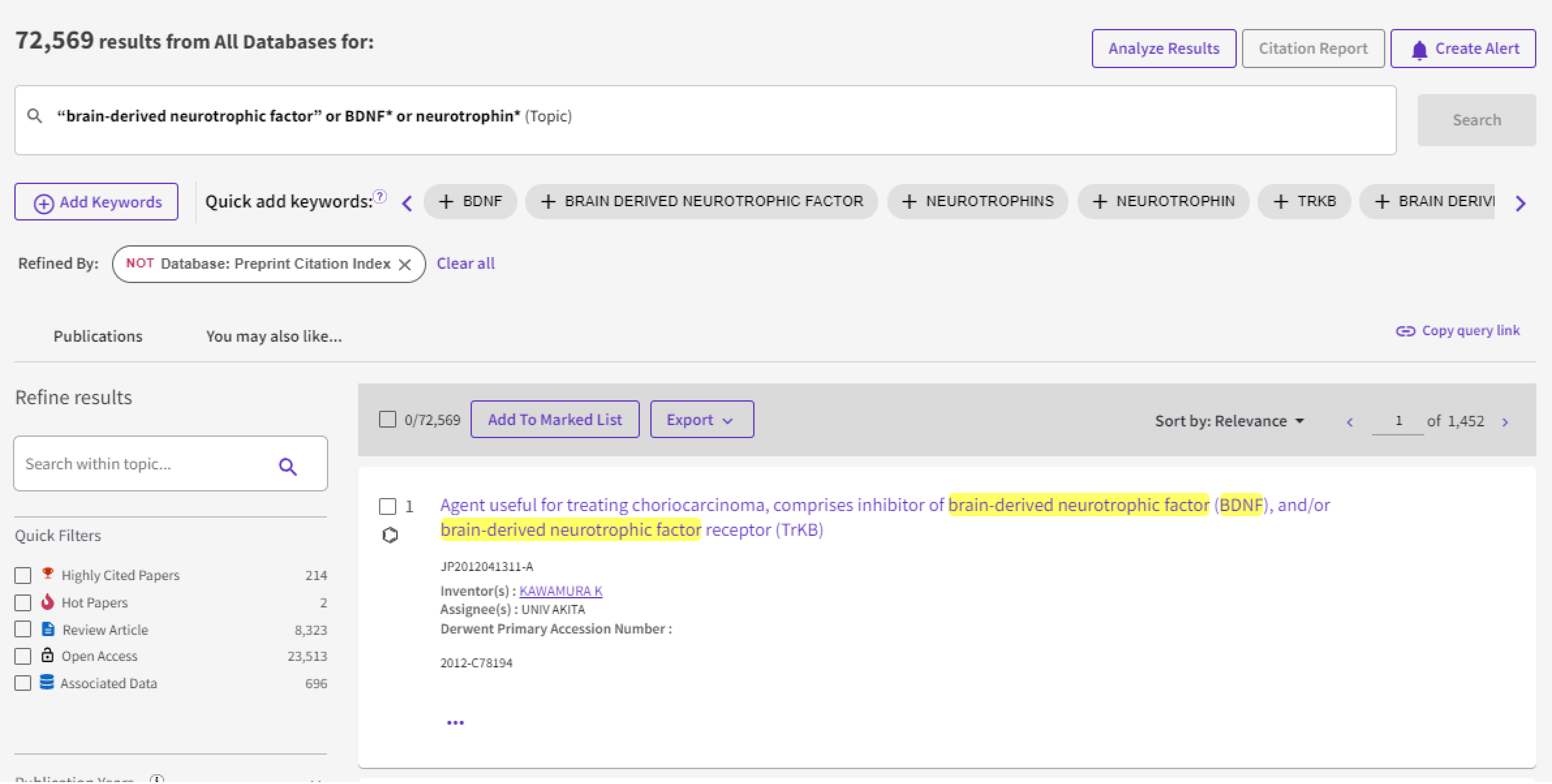
  more info...



Search in: All Databases

Collections: All

Topic: “brain-derived neurotrophic factor” or BDNF* or neurotrophin*



72,569 results from All Databases for:

"brain-derived neurotrophic factor" or BDNF* or neurotrophin* (Topic)

Quick add keywords: + BDNF + BRAIN DERIVED NEUROTROPHIC FACTOR + NEUROTROPHINS + NEUROTROPHIN + TRKB + BRAIN DERIVI

Refined By: NOT Database: Preprint Citation Index ✕

Refine results

Search within topic...

Quick Filters

☐	Highly Cited Papers	214
☐	Hot Papers	2
☐	Review Article	8,323
☐	Open Access	23,513
☐	Associated Data	696

☐ 0/72,569

Sort by: Relevance

1 of 1,452

☐ 1 Agent useful for treating choriocarcinoma, comprises inhibitor of brain-derived neurotrophic factor (BDNF), and/or brain-derived neurotrophic factor receptor (TrKB)

JP2012041311-A

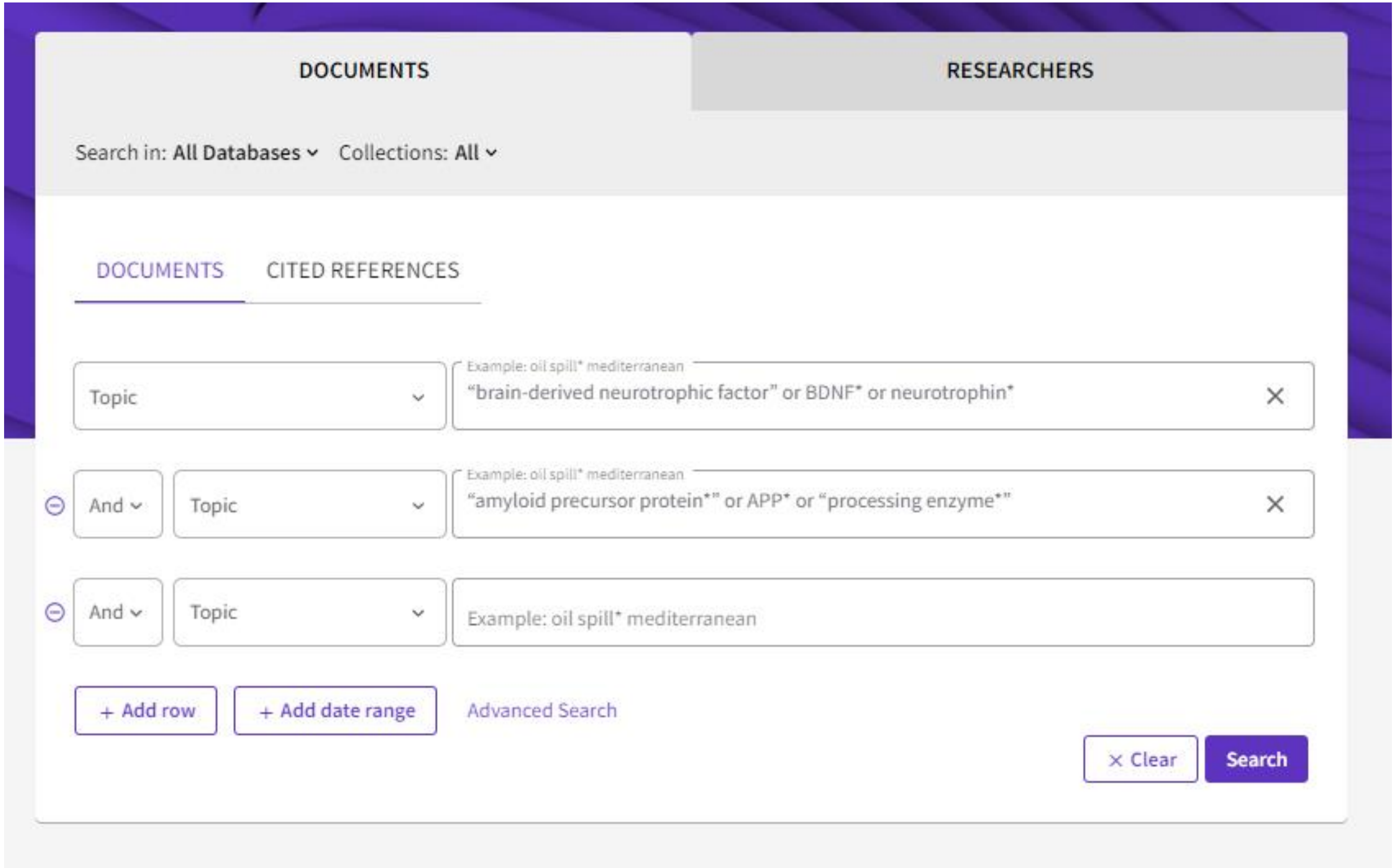
Inventor(s) : KAWAMURA K

Assignee(s) : UNIV AKITA

Derwent Primary Accession Number :

2012-C78194

...

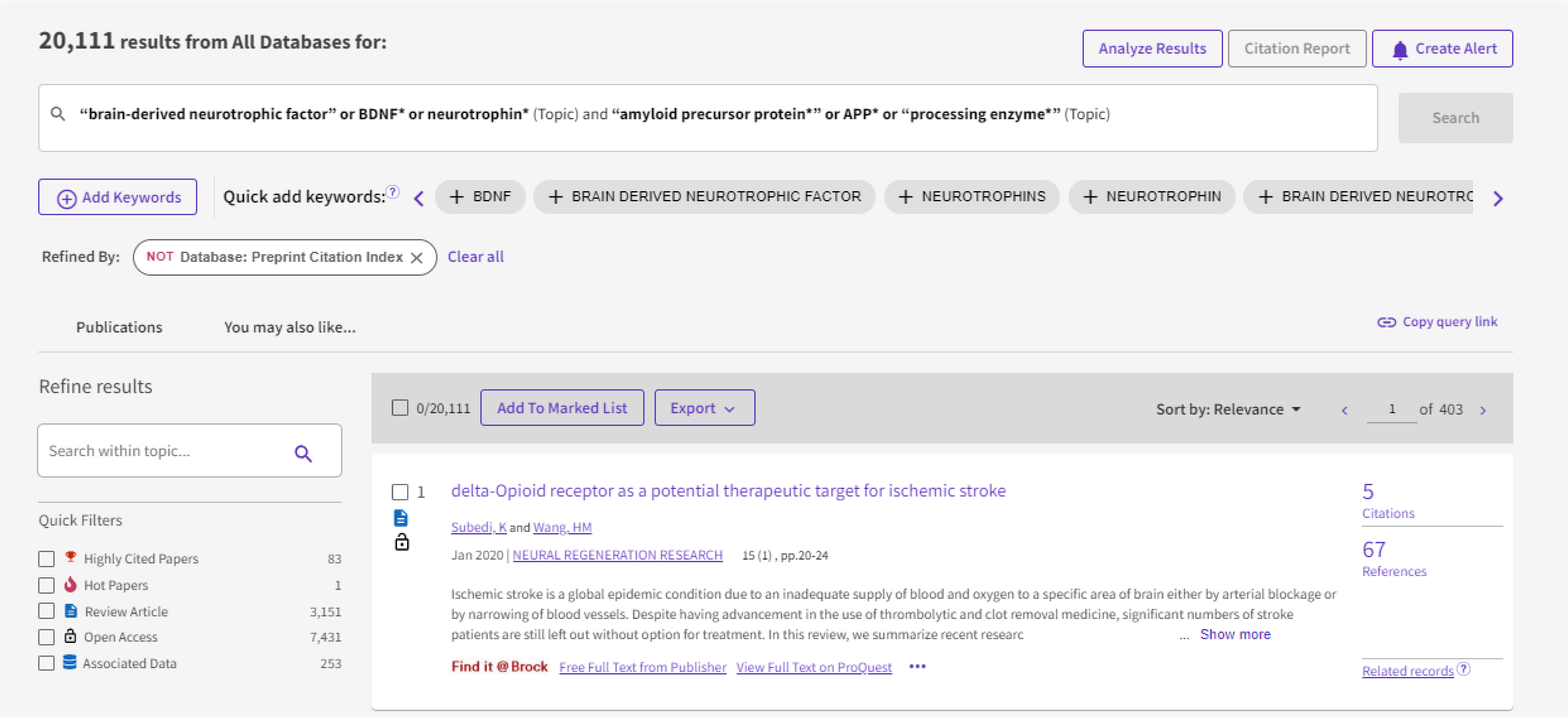


Search in: **All Databases** Collections: **All**

Topic: “brain-derived neurotrophic factor” or BDNF* or neurotrophin*

And Topic: “amyloid precursor protein*” or APP* or “processing enzyme*”

And Topic:



20,111 results from All Databases for:

"brain-derived neurotrophic factor" or BDNF* or neurotrophin* (Topic) and **"amyloid precursor protein*" or APP* or "processing enzyme*"** (Topic)

Refined By: NOT Database: Preprint Citation Index

Quick Filters

☐	Highly Cited Papers	83
☐	Hot Papers	1
☐	Review Article	3,151
☐	Open Access	7,431
☐	Associated Data	253

☐ 0/20,111

Sort by: Relevance

☐ 1 delta-Opioid receptor as a potential therapeutic target for ischemic stroke

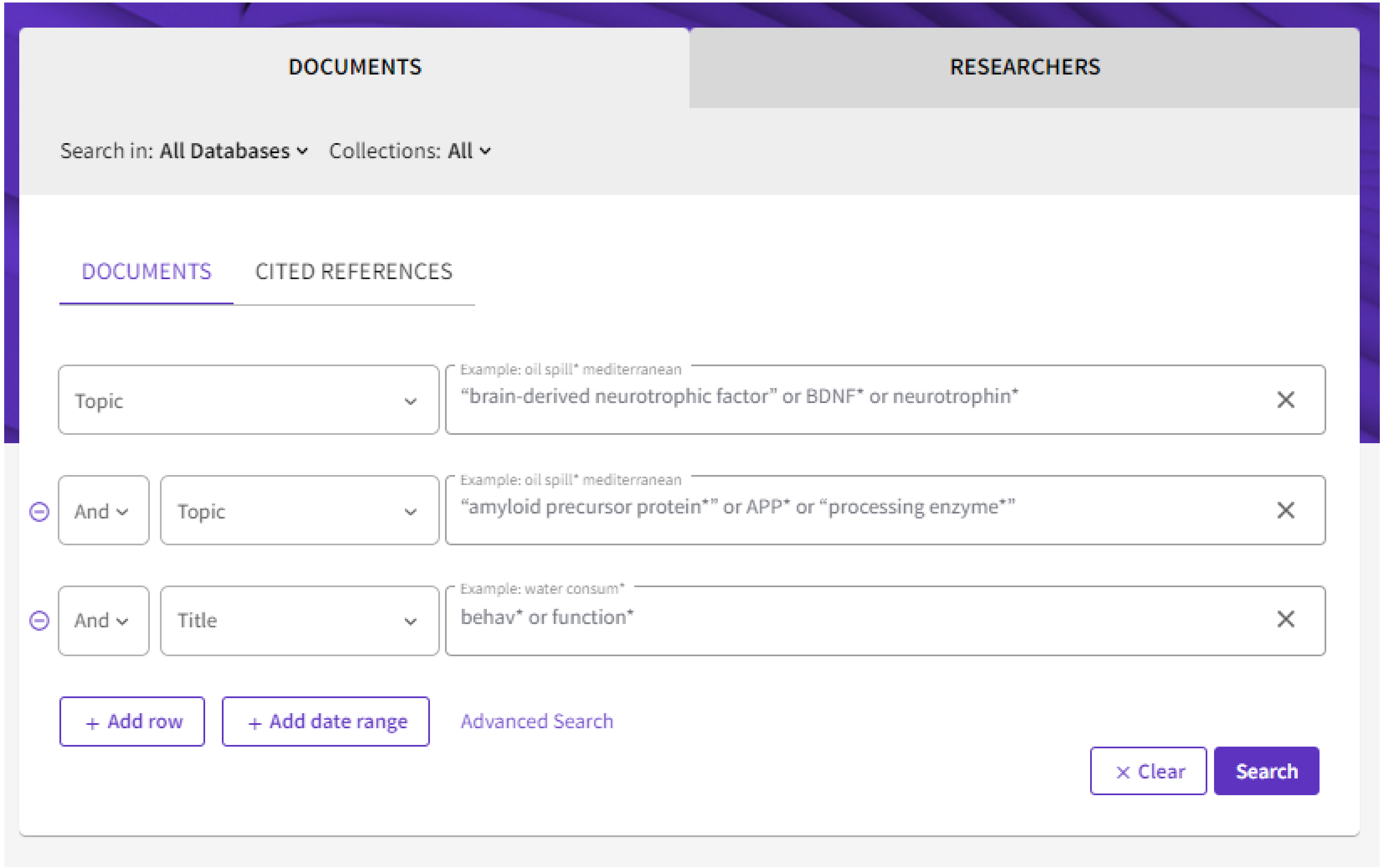
Subedi, K and Wang, HM

Jan 2020 | NEURAL REGENERATION RESEARCH 15 (1) , pp.20-24

Ischemic stroke is a global epidemic condition due to an inadequate supply of blood and oxygen to a specific area of brain either by arterial blockage or by narrowing of blood vessels. Despite having advancement in the use of thrombolytic and clot removal medicine, significant numbers of stroke patients are still left out without option for treatment. In this review, we summarize recent researc ... Show more

5 Citations

67 References

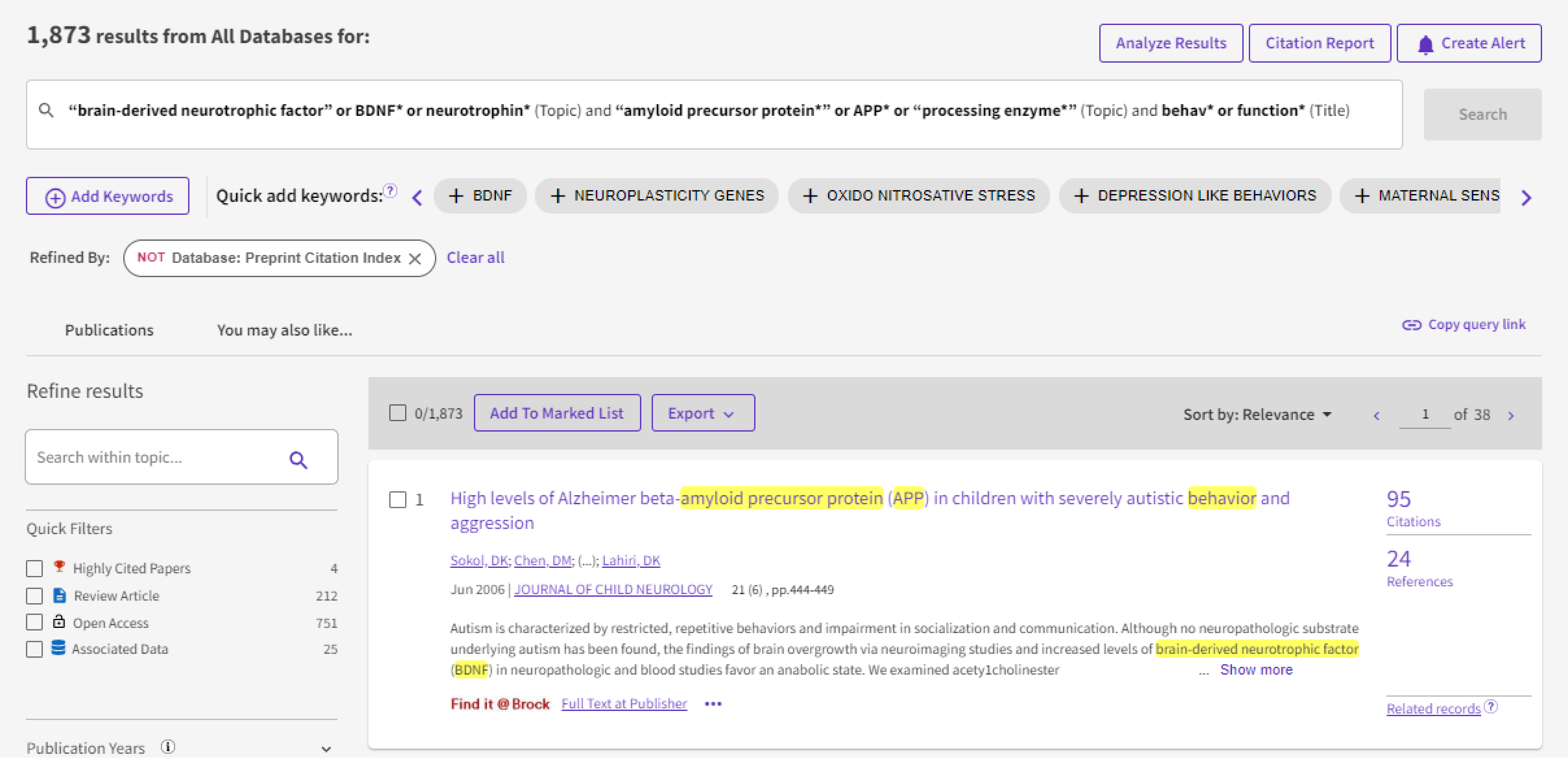


Search in: All Databases
Collections: All

Topic: “brain-derived neurotrophic factor” or BDNF* or neurotrophin*

And Topic: “amyloid precursor protein*” or APP* or “processing enzyme*”

And Title: behav* or function*



1,873 results from All Databases for:

“brain-derived neurotrophic factor” or BDNF* or neurotrophin* (Topic) and “amyloid precursor protein*” or APP* or “processing enzyme*” (Topic) and behav* or function* (Title)

Refined By: NOT Database: Preprint Citation Index

0/1,873

Sort by: Relevance

1 of 38

☐ 1 High levels of Alzheimer beta-amyloid precursor protein (APP) in children with severely autistic behavior and aggression

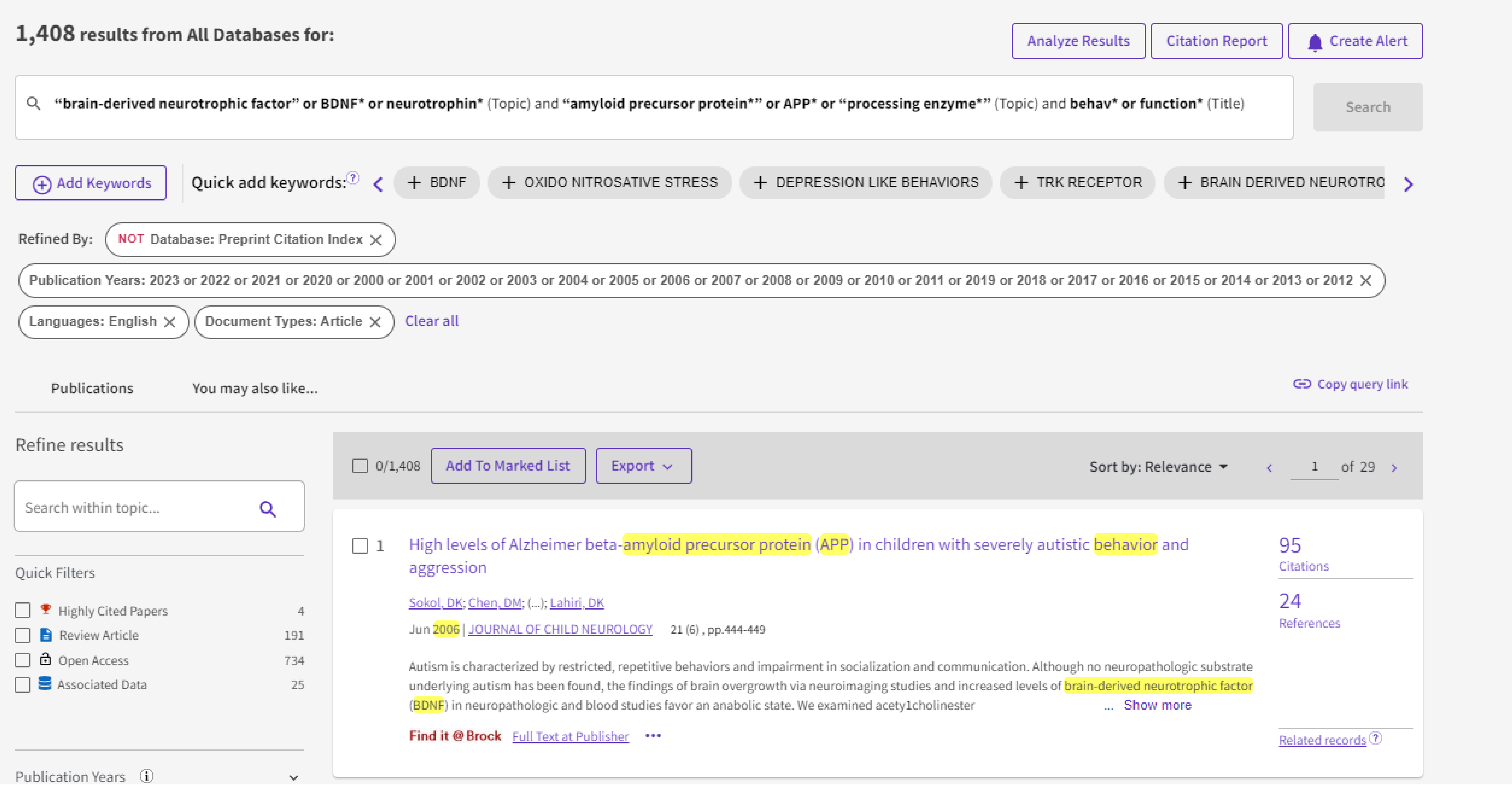
Sokol, DK; Chen, DM; (...); Lahiri, DK

Jun 2006 | JOURNAL OF CHILD NEUROLOGY 21 (6) , pp.444-449

Autism is characterized by restricted, repetitive behaviors and impairment in socialization and communication. Although no neuropathologic substrate underlying autism has been found, the findings of brain overgrowth via neuroimaging studies and increased levels of brain-derived neurotrophic factor (BDNF) in neuropathologic and blood studies favor an anabolic state. We examined acety1cholinester ... Show more

95 Citations

24 References



1,408 results from All Databases for:

“brain-derived neurotrophic factor” or BDNF* or neurotrophin* (Topic) and **“amyloid precursor protein*” or APP* or “processing enzyme*”** (Topic) and **behav* or function*** (Title)

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Publication Years: 2023 or 2022 or 2021 or 2020 or 2000 or 2001 or 2002 or 2003 or 2004 or 2005 or 2006 or 2007 or 2008 or 2009 or 2010 or 2011 or 2019 or 2018 or 2017 or 2016 or 2015 or 2014 or 2013 or 2012 ✕

Languages: English ✕ Document Types: Article ✕

Quick Filters

☐ Highly Cited Papers	4
☐ Review Article	191
☐ Open Access	734
☐ Associated Data	25

Publication Years

☐ 0/1,408

☐ 1 High levels of Alzheimer beta-amyloid precursor protein (APP) in children with severely autistic behavior and aggression

Sokol, DK; Chen, DM; (...); Lahiri, DK

Jun 2006 | JOURNAL OF CHILD NEUROLOGY 21 (6) , pp.444-449

Autism is characterized by restricted, repetitive behaviors and impairment in socialization and communication. Although no neuropathologic substrate underlying autism has been found, the findings of brain overgrowth via neuroimaging studies and increased levels of brain-derived neurotrophic factor (BDNF) in neuropathologic and blood studies favor an anabolic state. We examined acety1cholinester ... Show more

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APPLIED PHYSIOLOGY.

J Appl Physiol 135: 121–135, 2023.
First published June 1, 2023; doi:10.1152/japplphysiol.00114.2023

RESEARCH ARTICLE

Physical Activity and the Brain

Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

Bradley J. Baranowski,[1] Ahmad Mohammad,[1] Michael S. Finch,[1] Alex Brown,[1] Roopan Dhaliwal,[1] Daniel M. Marko,[1] Paul J. LeBlanc,[1] Cheryl M. McCormick,[2,4] Val A. Fajardo,[3,4] and Rebecca E. K. MacPherson[1,4]

[1]Department of Health Sciences, Brock University, St. Catharines, Ontario, Canada; [2]Department of Psychology, Brock University, St Catharines, Ontario, Canada; [3]Department of Kinesiology, Brock University, St Catharines, Ontario, Canada; and [4]Centre for Neuroscience, Brock University, St. Catharines, Ontario, Canada

Abstract

Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β-site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of Aβ. However, mechanisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth, and neuronal survival. Previously, our lab has shown using an ex vivo model that treatment of the prefrontal cortex with BDNF reduced BACE1 activity, highlighting a BDNF to BACE1 link. The purpose of this research was to examine whether BDNF treatments resulted in similar biochemical adaptations to APP processing as exercise training. Male C57BL6/J mice were assigned into one of four groups (n = 12/group): *1*) control; *2*) exercise training (progressive treadmill training 5 days/wk); *3*) BDNF (0.5 mg/kg body mass subcutaneous injection 5 days/wk); or *4*) endurance training and BDNF, for an 8-wk intervention. Recognition memory was measured with a novel object recognition test. Serum, the prefrontal cortex, and hippocampus were collected. BDNF improved recognition memory to a similar extent as endurance training. BDNF and exercise decreased BACE1 activity and increased ADAM10 activity in the prefrontal cortex, indicating a shift in APP processing. Our novel results indicate that BDNF exerts similar beneficial effects on cognition and APP processing as exercise training. Future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

NEW & NOTEWORTHY Our study presents the novel findings that chronic peripheral BDNF injections result in regulation of APP processing enzymes and improved cognition to a similar extent as exercise training. These findings highlight the potential efficacy of using BDNF as a therapeutic intervention in the prevention of neurodegenerative diseases (i.e., Alzheimer's disease). Furthermore, future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

amyloid precursor protein; BDNF; brain; cognition; exercise training

INTRODUCTION

The overproduction and accumulation of amyloid beta (Aβ) peptides that can result in plaque formation is a key feature of neurodegenerative diseases, such as Alzheimer's disease (AD). Aβ peptides have been shown to lead to neuronal loss, cognitive impairments, neuroinflammation, synaptic dysfunction, and other neurotoxic events in the brain (1). Alternatively, a lifelong underproduction of Aβ, as observed in individuals with the Icelandic mutation in amyloid precursor protein (APP), protects individuals from cognitive decline with aging (2). Increased production of these peptides can be observed decades before clinical diagnosis (3–6), highlighting the importance of early, preventative measures to reduce the Aβ burden as one ages. Aβ originates from a large transmembrane protein, known as the APP (7, 8). The fate of APP processing is determined by one of two competing pathways: the amyloidogenic and nonamyloidogenic pathways (9). The amyloidogenic pathway is initiated when the rate-limiting enzyme, β-site amyloid precursor protein cleaving enzyme 1 (BACE1), cleaves APP resulting in the release of a soluble ectodomain, soluble APPβ (sAPPβ) fragment, and leaving behind a membrane-bound C-terminal fragment (CTF) 89/99 (10, 11). ϒ-Secretase then cleaves the CTF89/99 fragment and releases

Correspondence: R. E. K. MacPherson (rmacpherson@brocku.ca).
Submitted 22 February 2023 / Revised 24 May 2023 / Accepted 25 May 2023

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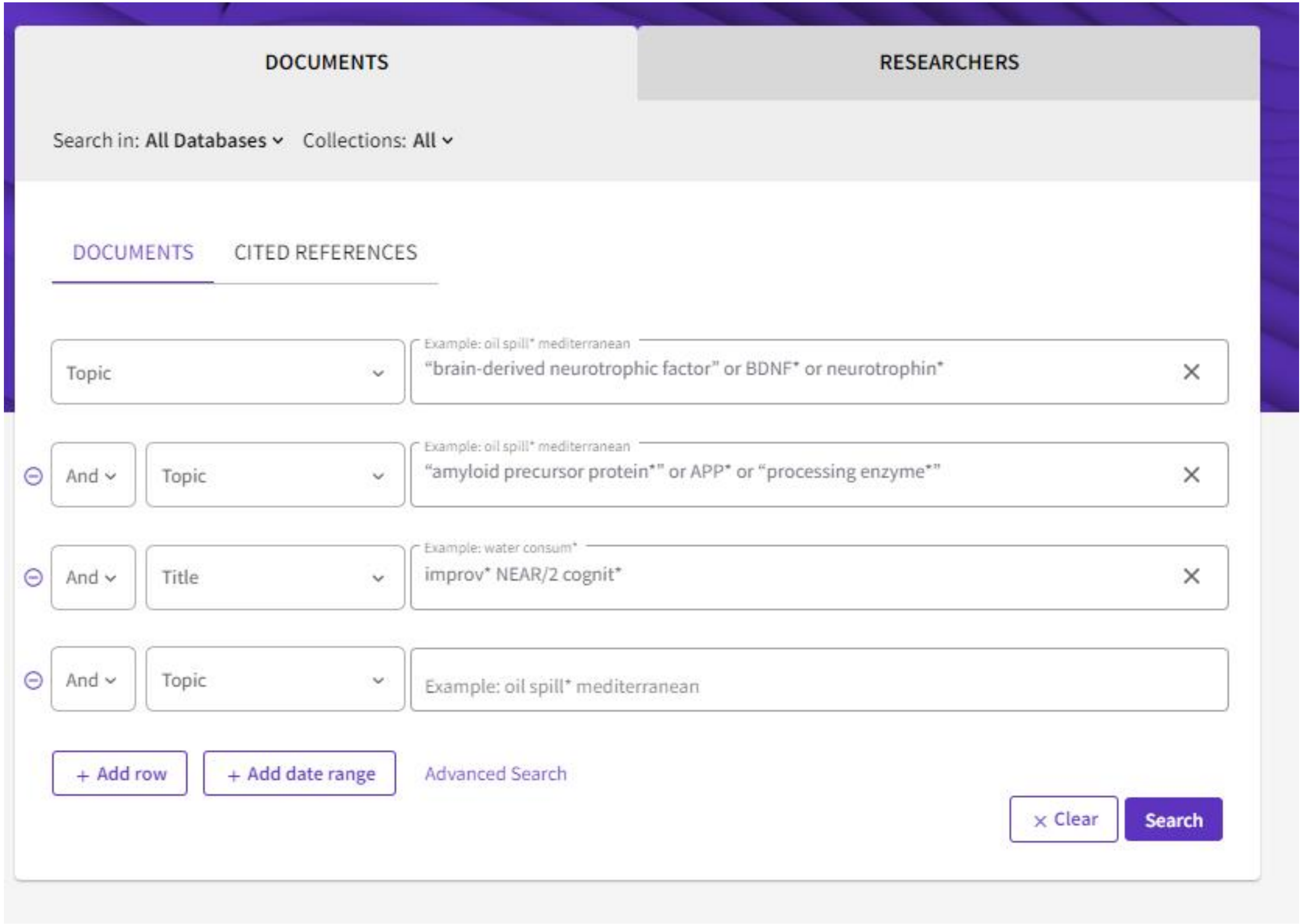
The purpose of this study was to determine if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements. We further examined if BDNF and endurance training in combination would result in a cumulative effect on APP processing. We hypothesize that BDNF treatment will result in similar behavioral and biochemical adaptations as endurance training as it relates to cognition and APP processing. The results from this study provide evidence in support of BDNF as an exercise mimetic that beneficially alters APP processing.



Search in: All Databases

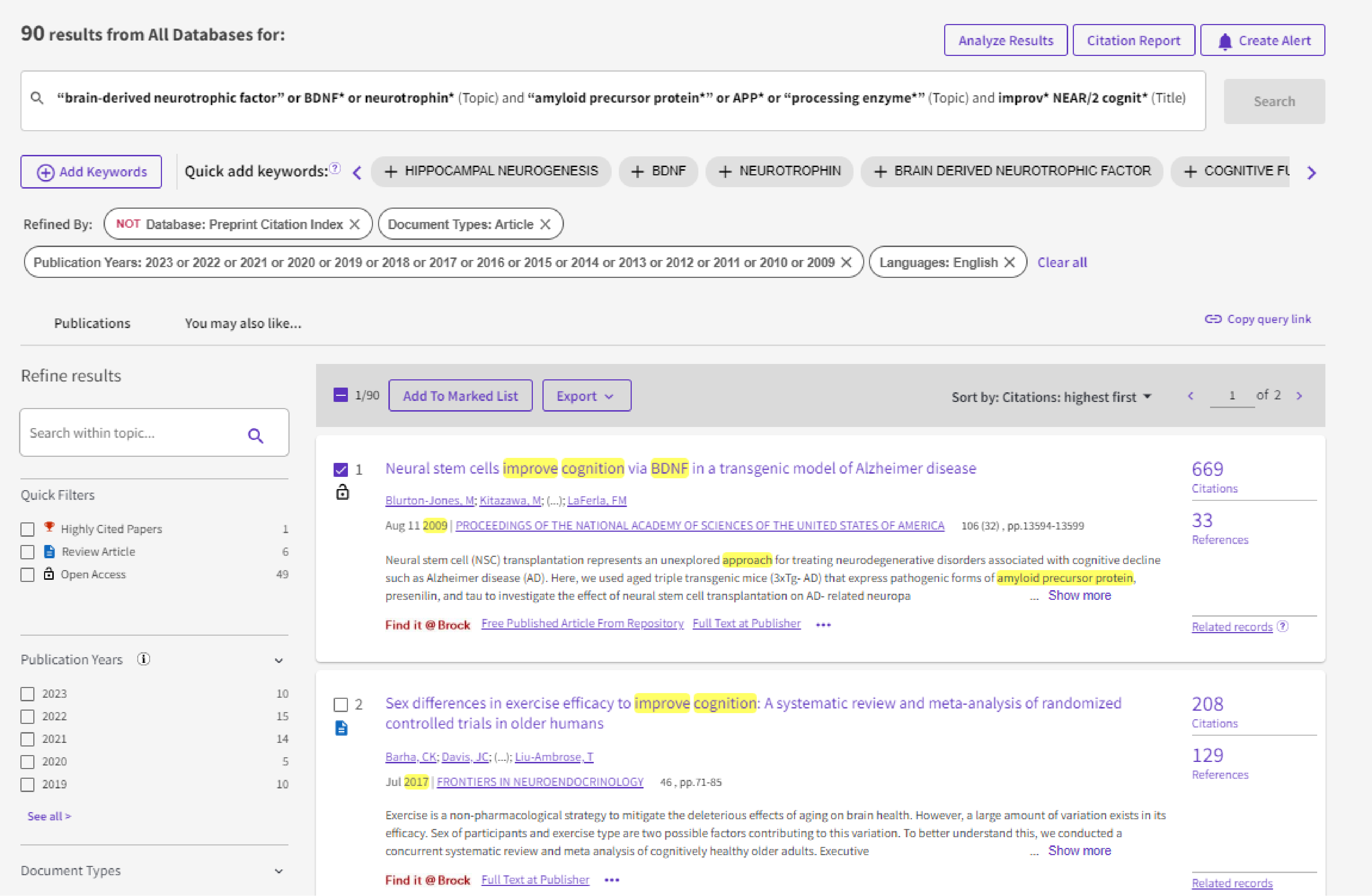
Collections: All

Topic: “brain-derived neurotrophic factor” or BDNF* or neurotrophin*

And Topic: “amyloid precursor protein*” or APP* or “processing enzyme*”

And Title: improv* NEAR/2 cognit*

And Topic:



90 results from All Databases for:

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☐ Review Article	6
☐ Open Access	49

Publication Years

☐ 2023	10
☐ 2022	15
☐ 2021	14
☐ 2020	5
☐ 2019	10

☐ 1/90

Sort by: Citations: highest first

1 of 2

☑ 1

Neural stem cells improve cognition via BDNF in a transgenic model of Alzheimer disease

Blurton-Jones, M; Kitazawa, M; (...); LaFerla, FM

Aug 11 2009 | PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA 106 (32) , pp.13594-13599

Neural stem cell (NSC) transplantation represents an unexplored approach for treating neurodegenerative disorders associated with cognitive decline such as Alzheimer disease (AD). Here, we used aged triple transgenic mice (3xTg- AD) that express pathogenic forms of amyloid precursor protein, presenilin, and tau to investigate the effect of neural stem cell transplantation on AD- related neuropa ... Show more

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669 Citations

33 References

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Sex differences in exercise efficacy to improve cognition: A systematic review and meta-analysis of randomized controlled trials in older humans

Barha, CK; Davis, JC; (...); Liu-Ambrose, T

Jul 2017 | FRONTIERS IN NEUROENDOCRINOLOGY 46 , pp.71-85

Exercise is a non-pharmacological strategy to mitigate the deleterious effects of aging on brain health. However, a large amount of variation exists in its efficacy. Sex of participants and exercise type are two possible factors contributing to this variation. To better understand this, we conducted a concurrent systematic review and meta analysis of cognitively healthy older adults. Executive ... Show more

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129 References

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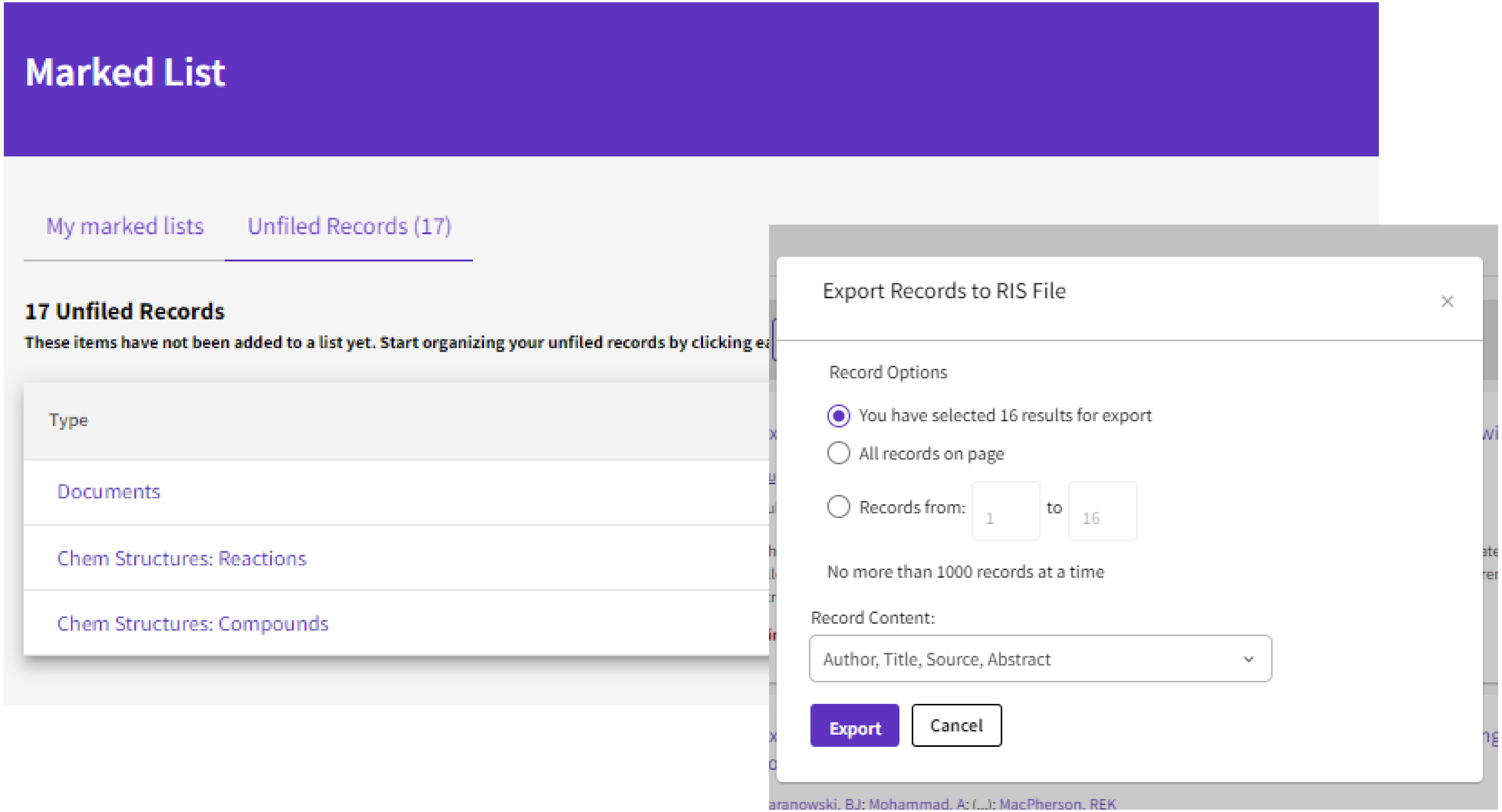

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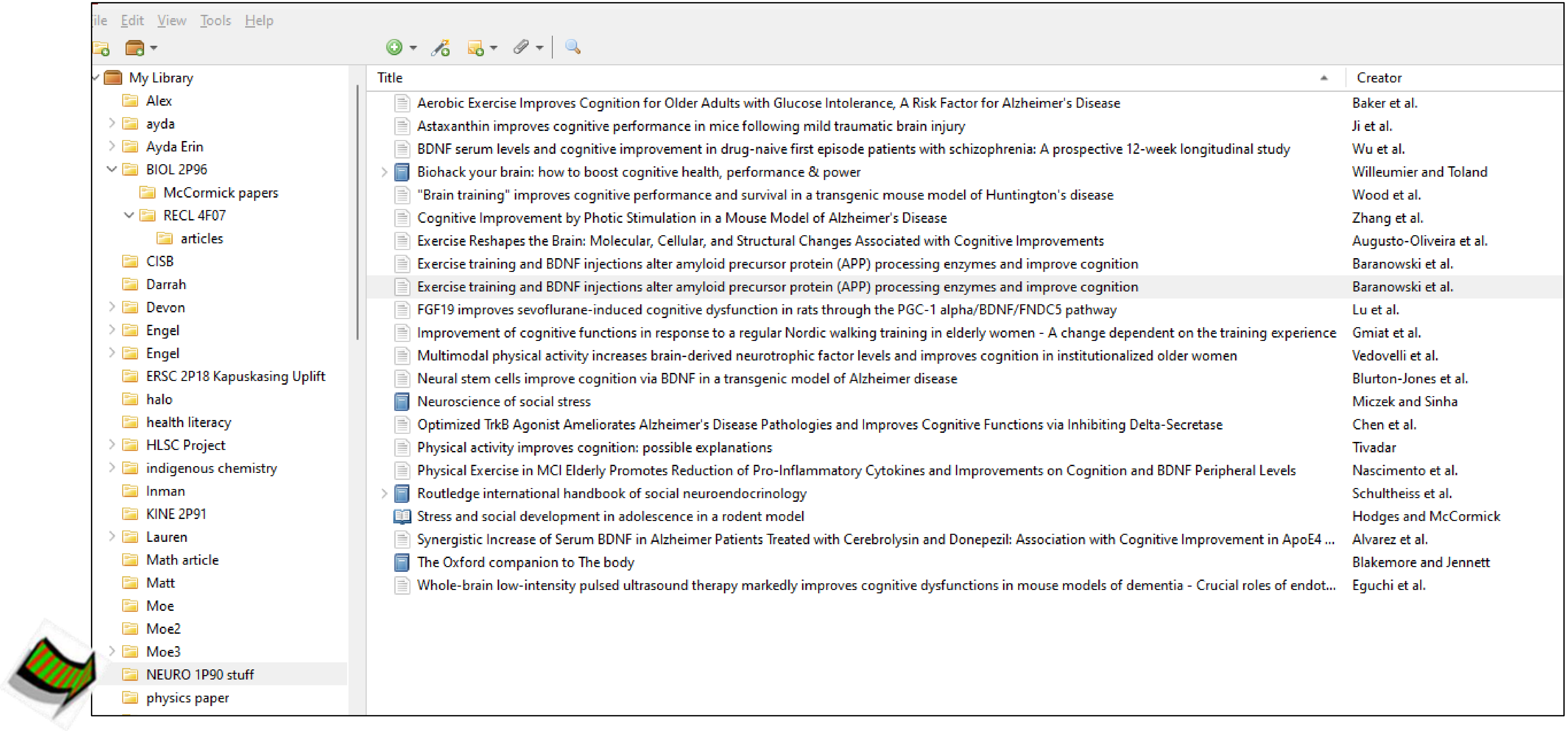

Title	Creator
Aerobic Exercise Improves Cognition for Older Adults with Glucose Intolerance, A Risk Factor for Alzheimer's Disease	Baker et al.
Astaxanthin improves cognitive performance in mice following mild traumatic brain injury	Ji et al.
BDNF serum levels and cognitive improvement in drug-naive first episode patients with schizophrenia: A prospective 12-week longitudinal study	Wu et al.
Biohack your brain: how to boost cognitive health, performance & power	Willeumier and Toland
"Brain training" improves cognitive performance and survival in a transgenic mouse model of Huntington's disease	Wood et al.
Cognitive Improvement by Photic Stimulation in a Mouse Model of Alzheimer's Disease	Zhang et al.
Exercise Reshapes the Brain: Molecular, Cellular, and Structural Changes Associated with Cognitive Improvements	Augusto-Oliveira et al.
Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition	Baranowski et al.
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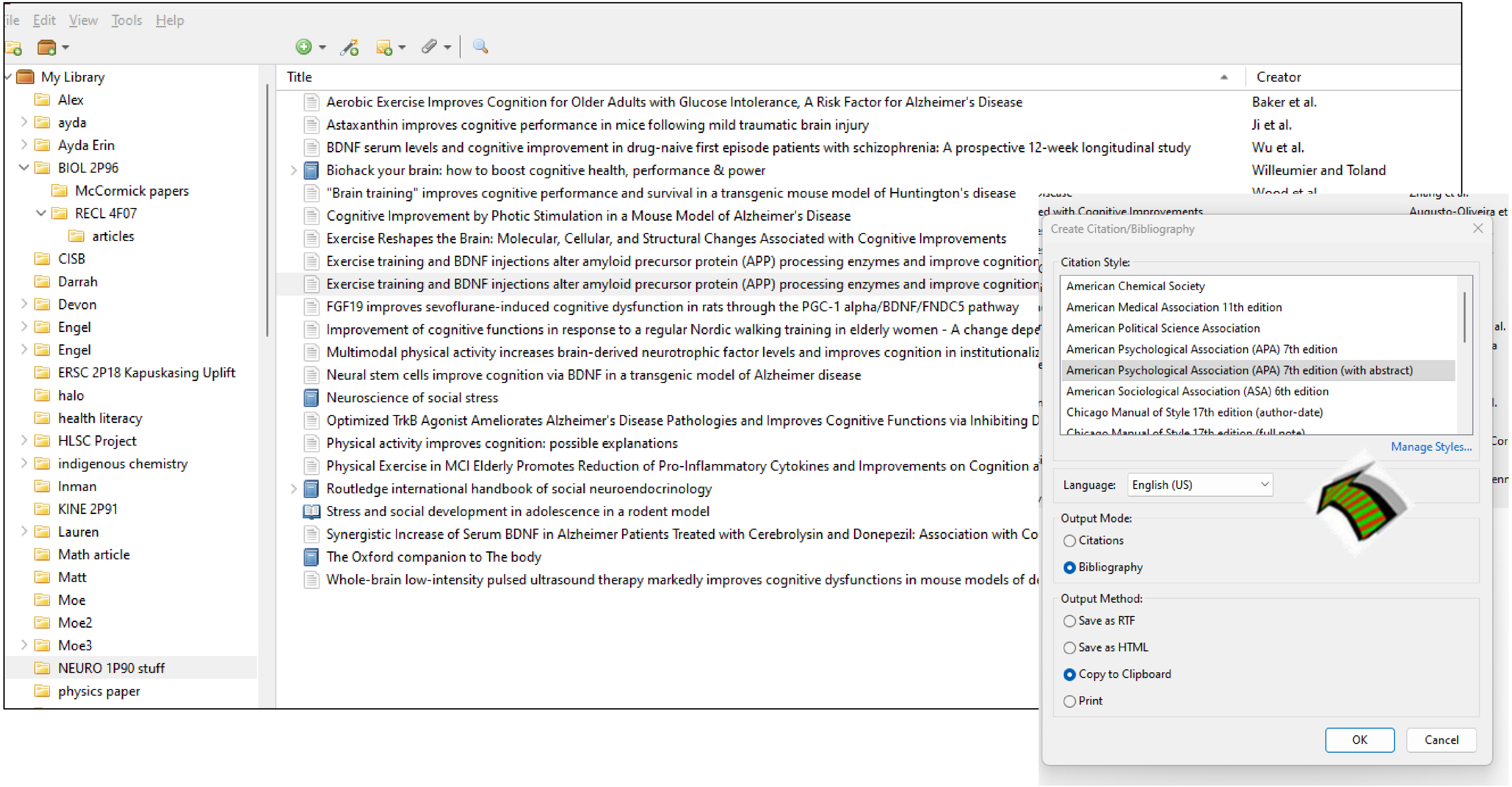
FGF19 improves sevoflurane-induced cognitive dysfunction in rats through the PGC-1 alpha/BDNF/FNDC5 pathway	Lu et al.
Improvement of cognitive functions in response to a regular Nordic walking training in elderly women - A change dependent on the training experience	Gmiat et al.
Multimodal physical activity increases brain-derived neurotrophic factor levels and improves cognition in institutionalized older women	Vedovelli et al.
Neural stem cells improve cognition via BDNF in a transgenic model of Alzheimer disease	Blurton-Jones et al.
Neuroscience of social stress	Miczek and Sinha
Optimized TrkB Agonist Ameliorates Alzheimer's Disease Pathologies and Improves Cognitive Functions via Inhibiting Delta-Secretase	Chen et al.
Physical activity improves cognition: possible explanations	Tivadar
Physical Exercise in MCI Elderly Promotes Reduction of Pro-Inflammatory Cytokines and Improvements on Cognition and BDNF Peripheral Levels	Nascimento et al.
Routledge international handbook of social neuroendocrinology	Schultheiss et al.
Stress and social development in adolescence in a rodent model	Hodges and McCormick
Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Cognitive Improvement in ApoE4 ...	Alvarez et al.
The Oxford companion to The body	Blakemore and Jennett
Whole-brain low-intensity pulsed ultrasound therapy markedly improves cognitive dysfunctions in mouse models of dementia - Crucial roles of endot...	Eguchi et al.



Title	Creator
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Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Co	
The Oxford companion to The body	
Whole-brain low-intensity pulsed ultrasound therapy markedly improves cognitive dysfunctions in mouse models of de	

Create Citation/Bibliography

Citation Style:

- American Chemical Society
- American Medical Association 11th edition
- American Political Science Association
- American Psychological Association (APA) 7th edition
- American Psychological Association (APA) 7th edition (with abstract)
- American Sociological Association (ASA) 6th edition
- Chicago Manual of Style 17th edition (author-date)
- Chicago Manual of Style 17th edition (full note)

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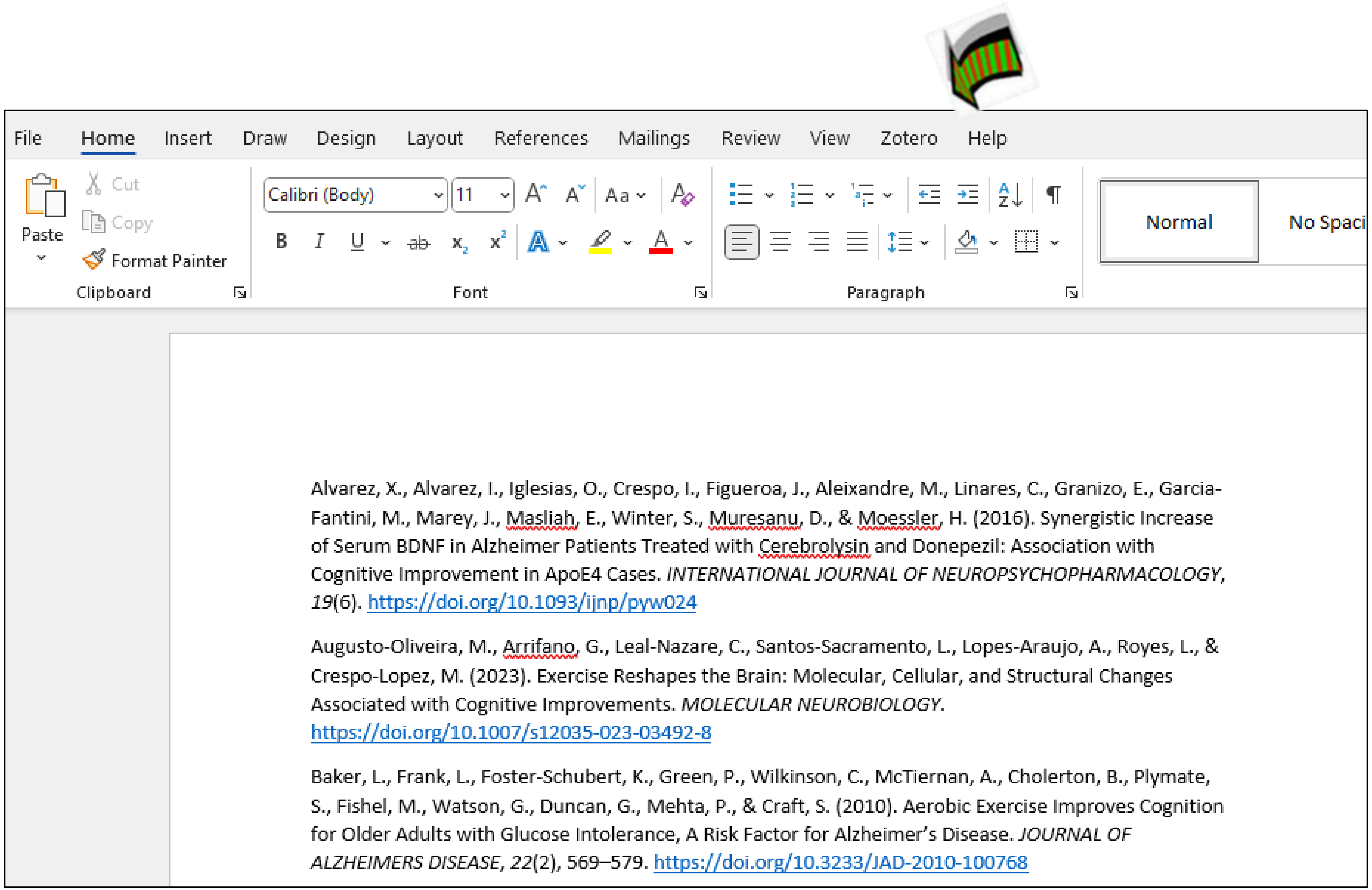
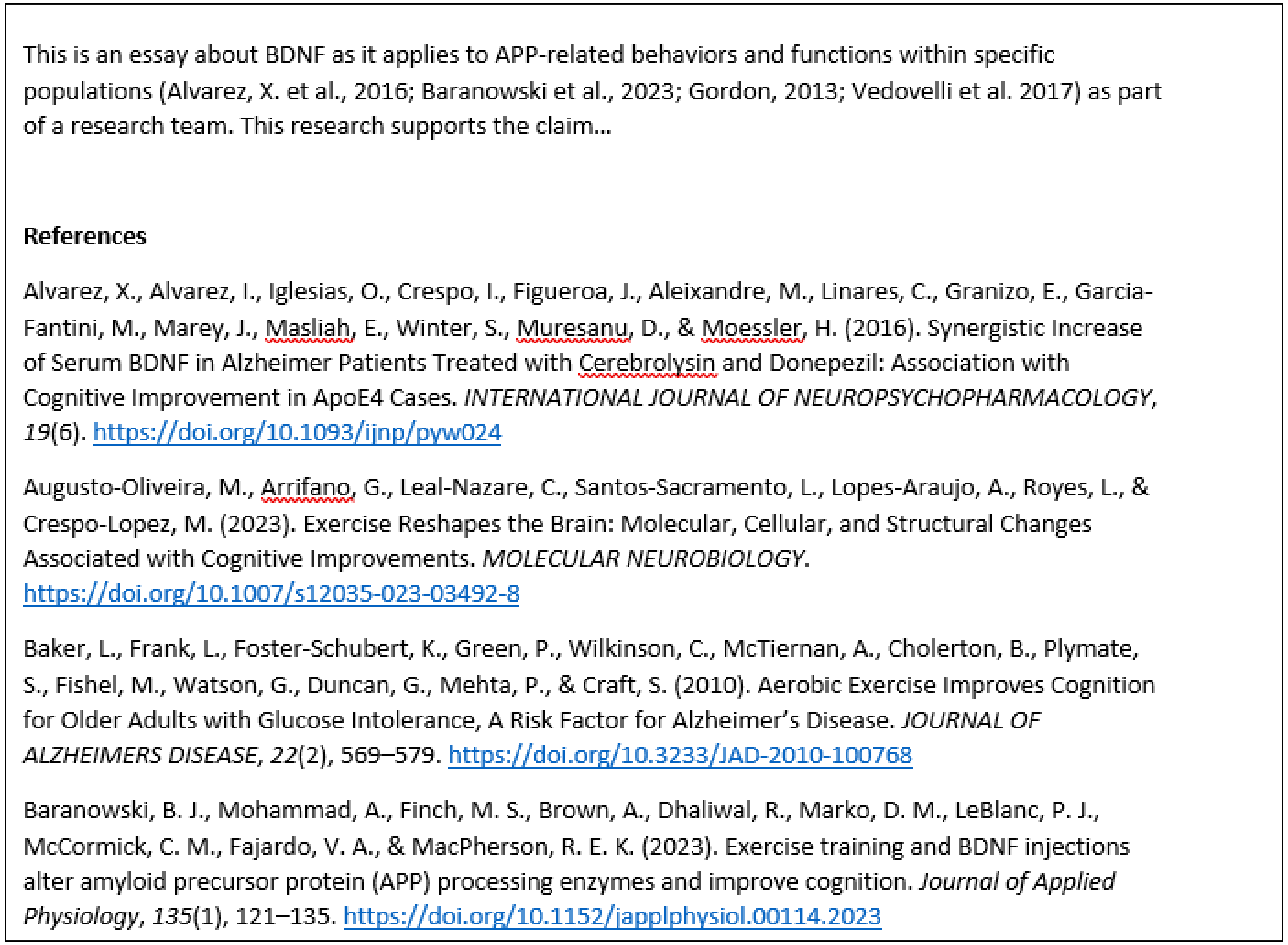


Alvarez, X., Alvarez, I., Iglesias, O., Crespo, I., Figueroa, J., Aleixandre, M., Linares, C., Granizo, E., Garcia-Fantini, M., Marey, J., Masliah, E., Winter, S., Muresanu, D., & Moessler, H. (2016). Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Cognitive Improvement in ApoE4 Cases. *INTERNATIONAL JOURNAL OF NEUROPSYCHOPHARMACOLOGY*, *19*(6). https://doi.org/10.1093/ijnp/pyw024

Augusto-Oliveira, M., Arrifano, G., Leal-Nazare, C., Santos-Sacramento, L., Lopes-Araujo, A., Royes, L., & Crespo-Lopez, M. (2023). Exercise Reshapes the Brain: Molecular, Cellular, and Structural Changes Associated with Cognitive Improvements. *MOLECULAR NEUROBIOLOGY*. https://doi.org/10.1007/s12035-023-03492-8

Baker, L., Frank, L., Foster-Schubert, K., Green, P., Wilkinson, C., McTiernan, A., Cholerton, B., Plymate, S., Fishel, M., Watson, G., Duncan, G., Mehta, P., & Craft, S. (2010). Aerobic Exercise Improves Cognition for Older Adults with Glucose Intolerance, A Risk Factor for Alzheimer’s Disease. *JOURNAL OF ALZHEIMERS DISEASE*, *22*(2), 569–579. https://doi.org/10.3233/JAD-2010-100768



This is an essay about BDNF as it applies to APP-related behaviors and functions within specific populations (Alvarez, X. et al., 2016; Baranowski et al., 2023; Gordon, 2013; Vedovelli et al. 2017) as part of a research team. This research supports the claim…

References

Alvarez, X., Alvarez, I., Iglesias, O., Crespo, I., Figueroa, J., Aleixandre, M., Linares, C., Granizo, E., Garcia-Fantini, M., Marey, J., Masliah, E., Winter, S., Muresanu, D., & Moessler, H. (2016). Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Cognitive Improvement in ApoE4 Cases. *INTERNATIONAL JOURNAL OF NEUROPSYCHOPHARMACOLOGY*, *19*(6). https://doi.org/10.1093/ijnp/pyw024

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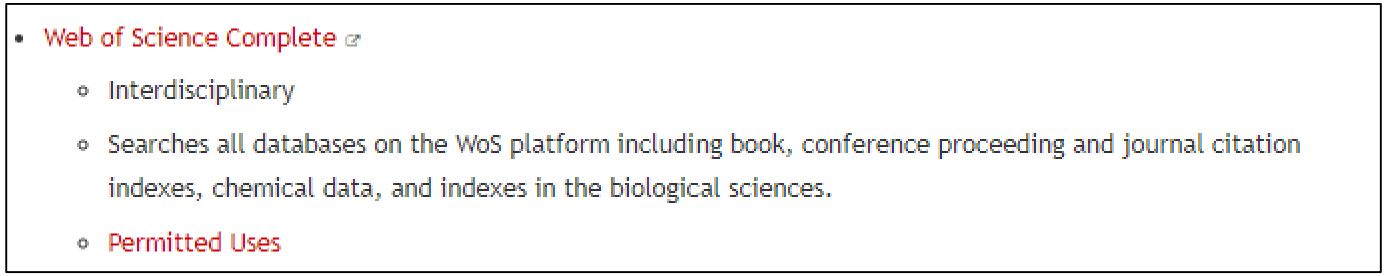
Baranowski, B. J., Mohammad, A., Finch, M. S., Brown, A., Dhaliwal, R., Marko, D. M., LeBlanc, P. J., McCormick, C. M., Fajardo, V. A., & MacPherson, R. E. K. (2023). Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition. *Journal of Applied Physiology*, *135*(1), 121–135. https://doi.org/10.1152/japplphysiol.00114.2023

Your turn!

How many scholarly citations of interest can you find in the Web of Science Complete Database on an essay topic “Controversies of the use of eponyms in neurosciences” for a class seminar on this topic?

Scholarly articles, English Language, 2000+, with citations and cited by references?

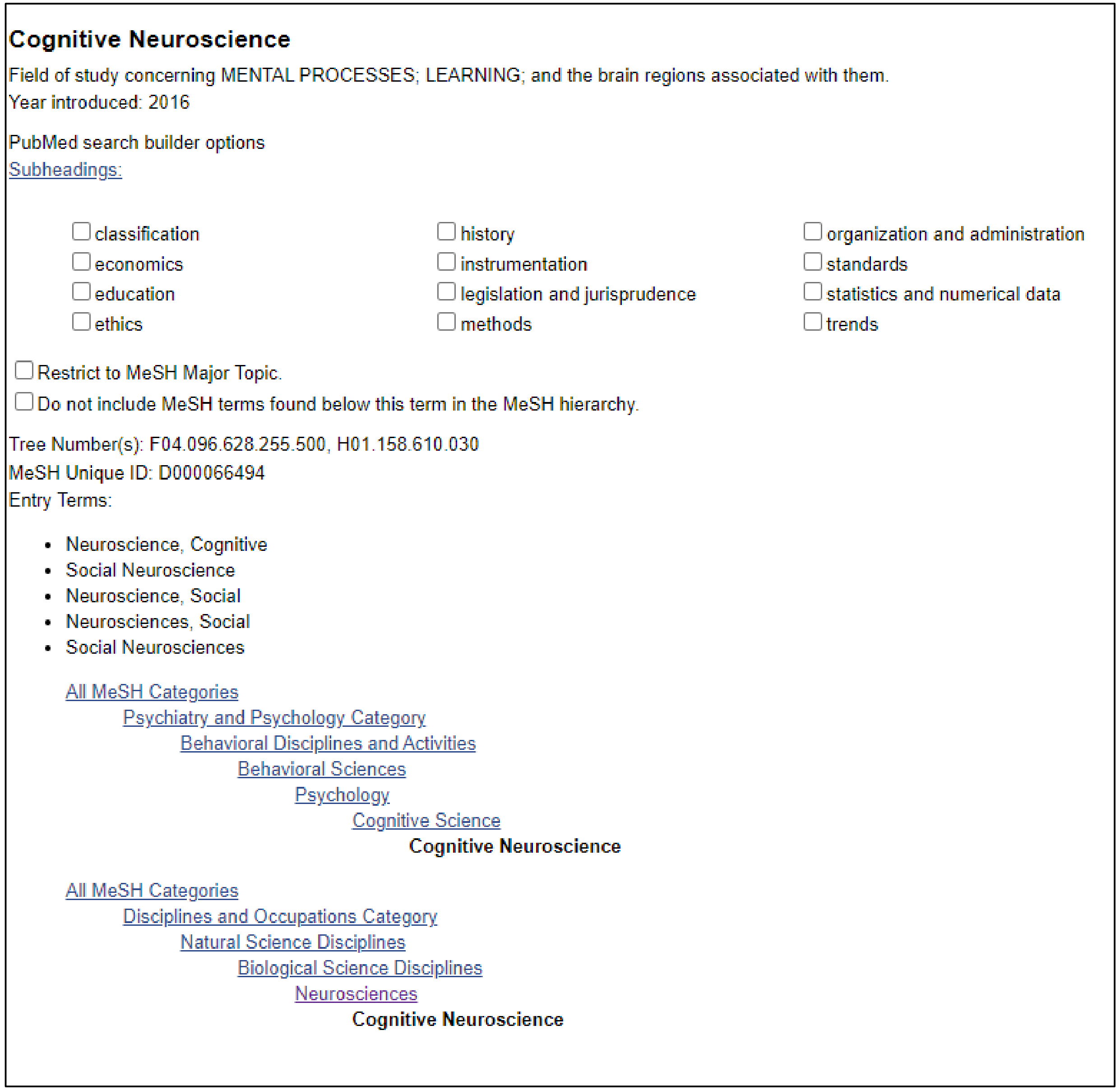


- Web of Science Complete
    - Interdisciplinary
    - Searches all databases on the WoS platform including book, conference proceeding and journal citation indexes, chemical data, and indexes in the biological sciences.
    - Permitted Uses

MeSH Cognitive Neuroscience
https://www.ncbi.nlm.nih.gov/mesh/2009646

MeSH Neurology
https://www.ncbi.nlm.nih.gov/mesh/68009462

MeSH Eponyms
https://www.ncbi.nlm.nih.gov/mesh/68004850



Cognitive Neuroscience

Field of study concerning MENTAL PROCESSES; LEARNING; and the brain regions associated with them.
Year introduced: 2016

PubMed search builder options
Subheadings:

☐ classification
☐ economics
☐ education
☐ ethics
☐ history
☐ instrumentation
☐ legislation and jurisprudence
☐ methods
☐ organization and administration
☐ standards
☐ statistics and numerical data
☐ trends

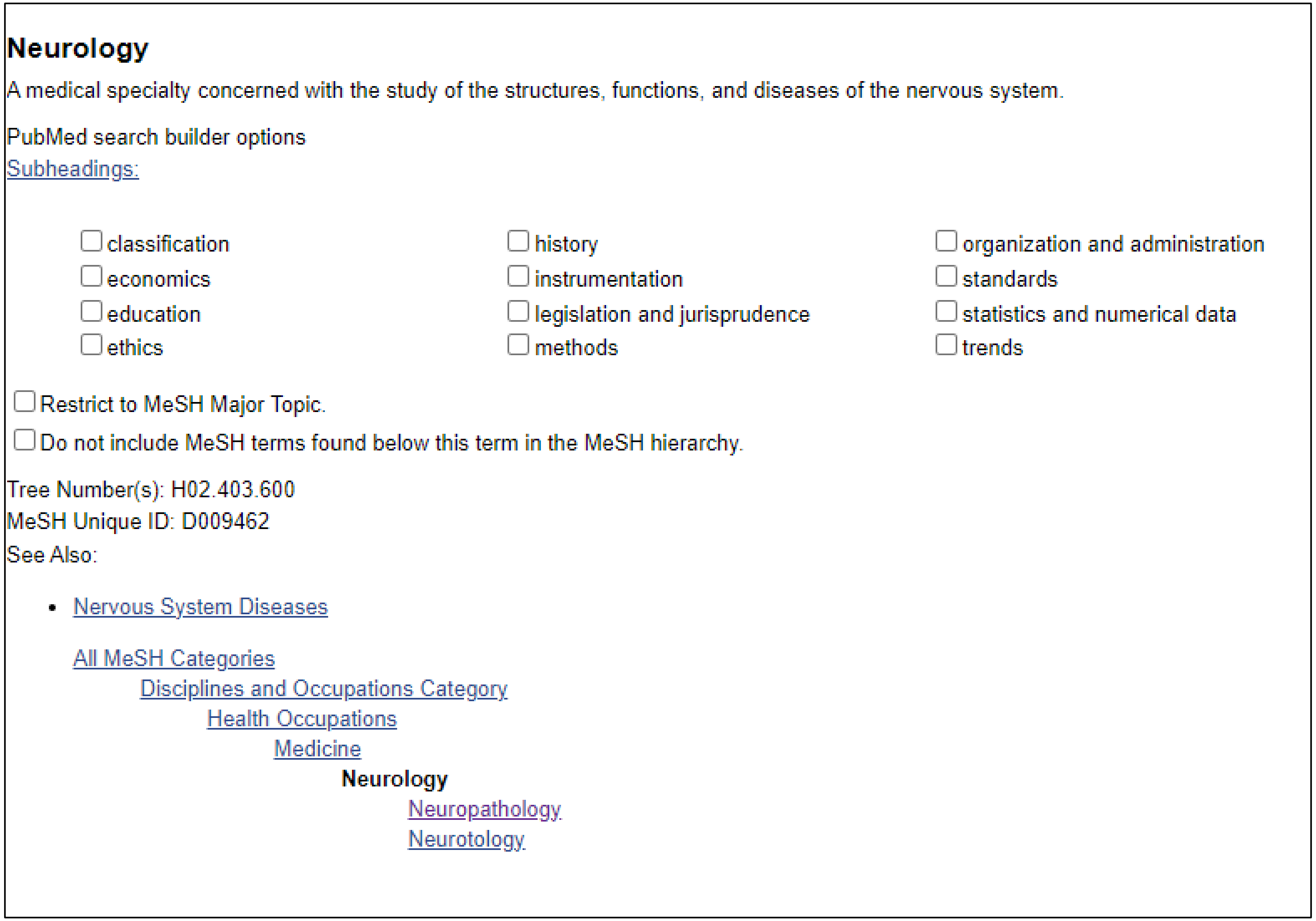
☐ Restrict to MeSH Major Topic.
☐ Do not include MeSH terms found below this term in the MeSH hierarchy.

Tree Number(s): F04.096.628.255.500, H01.158.610.030
MeSH Unique ID: D000066494
Entry Terms:

- Neuroscience, Cognitive
- Social Neuroscience
- Neuroscience, Social
- Neurosciences, Social
- Social Neurosciences

All MeSH Categories
  Psychiatry and Psychology Category
    Behavioral Disciplines and Activities
      Behavioral Sciences
        Psychology
          Cognitive Science
            Cognitive Neuroscience

All MeSH Categories
  Disciplines and Occupations Category
    Natural Science Disciplines
      Biological Science Disciplines
        Neurosciences
          Cognitive Neuroscience



Neurology

A medical specialty concerned with the study of the structures, functions, and diseases of the nervous system.

PubMed search builder options
Subheadings:

☐ classification
☐ economics
☐ education
☐ ethics
☐ history
☐ instrumentation
☐ legislation and jurisprudence
☐ methods
☐ organization and administration
☐ standards
☐ statistics and numerical data
☐ trends

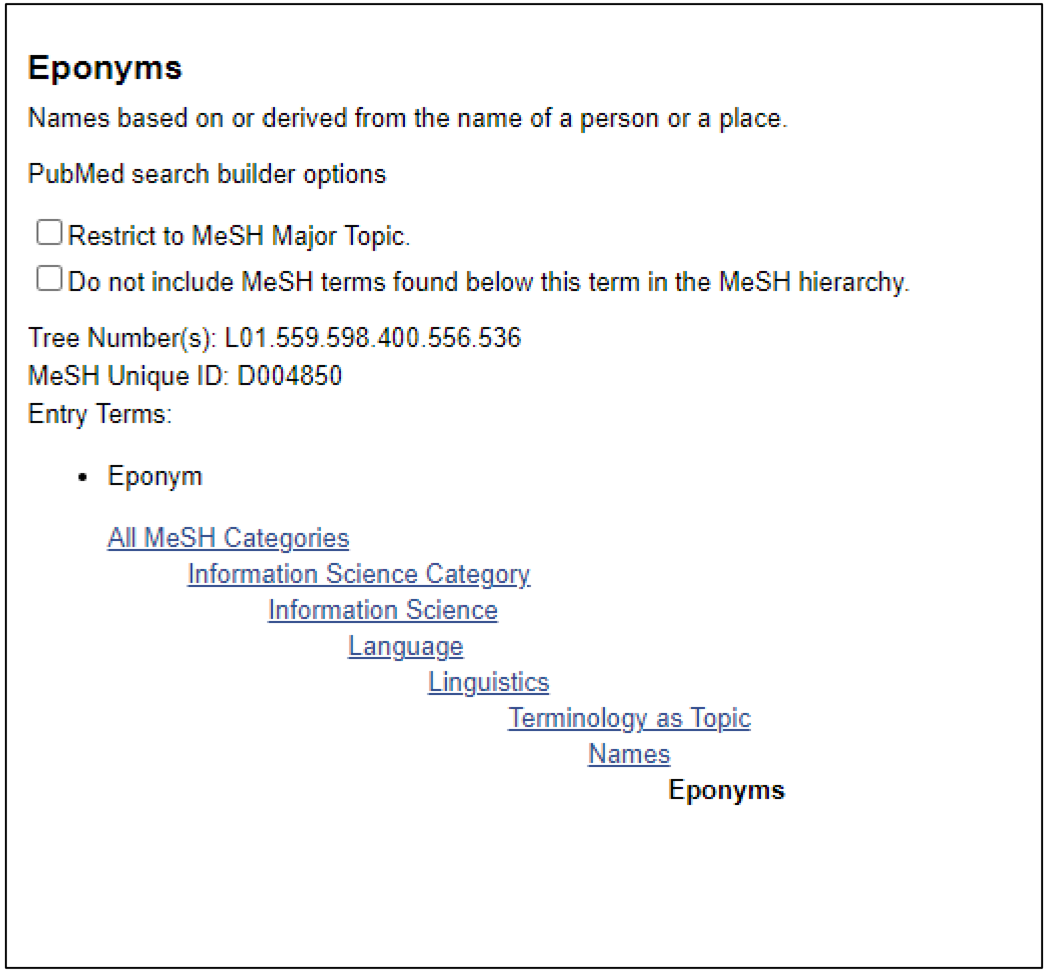
☐ Restrict to MeSH Major Topic.

☐ Do not include MeSH terms found below this term in the MeSH hierarchy.

Tree Number(s): H02.403.600
MeSH Unique ID: D009462
See Also:

- Nervous System Diseases

All MeSH Categories
  Disciplines and Occupations Category
    Health Occupations
      Medicine
        Neurology
          Neuropathology
          Neurotology



Eponyms

Names based on or derived from the name of a person or a place.

PubMed search builder options

☐ Restrict to MeSH Major Topic.

☐ Do not include MeSH terms found below this term in the MeSH hierarchy.

Tree Number(s): L01.559.598.400.556.536
MeSH Unique ID: D004850
Entry Terms:

- Eponym

All MeSH Categories
Information Science Category
Information Science
Language
Linguistics
Terminology as Topic
Names
Eponyms

Controversies on the use of eponyms in neurosciences.

Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers

Neuroscience tip and tricks

Look for seed articles
Identify main concepts
Look for alternate terms
Consult thesauri
Define terms
Create search strategies
Select database(s)
Think like the database when searching
Search, refine, filter and browse…
Cite, send to Zotero
Refine

Never too early to start
Be open to change
Ask for assistance
Be done!

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- Questions / Answers

Where can I get help?

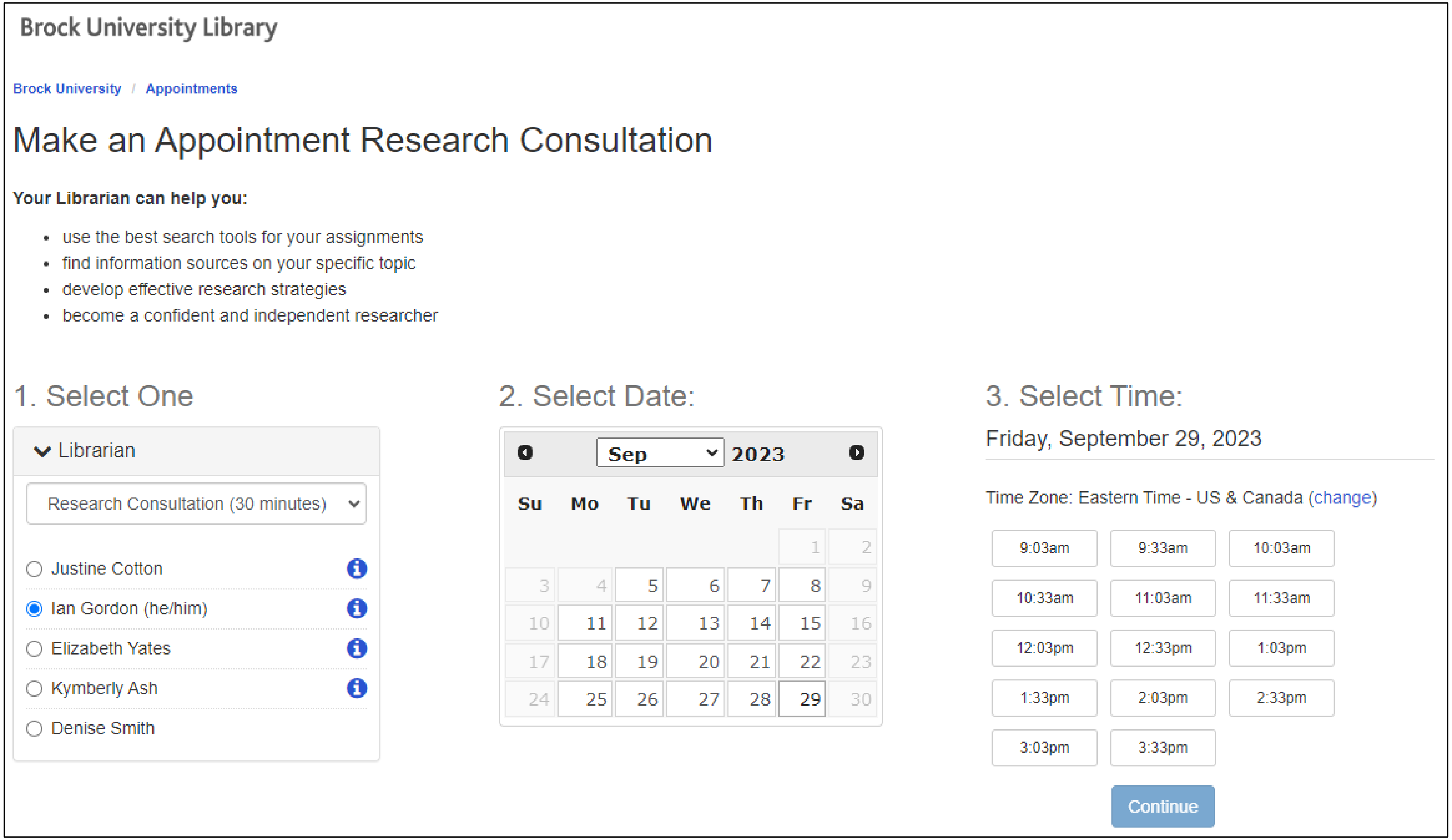
Brock Library Neuroscience Research Guide
https://researchguides.library.brocku.ca/NEUR

Email the Library
libhelp@brocku.ca

Ask Us Chat service
https://brocku.ca/library/chat/

Ask Us Chat

Book a Consultation
https://calendar.library.brocku.ca/appointments/researchconsultation



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- find information sources on your specific topic
- develop effective research strategies
- become a confident and independent researcher

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Librarian

Research Consultation (30 minutes)

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● Ian Gordon (he/him)
○ Elizabeth Yates
○ Kymberly Ash
○ Denise Smith

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Sep 2023

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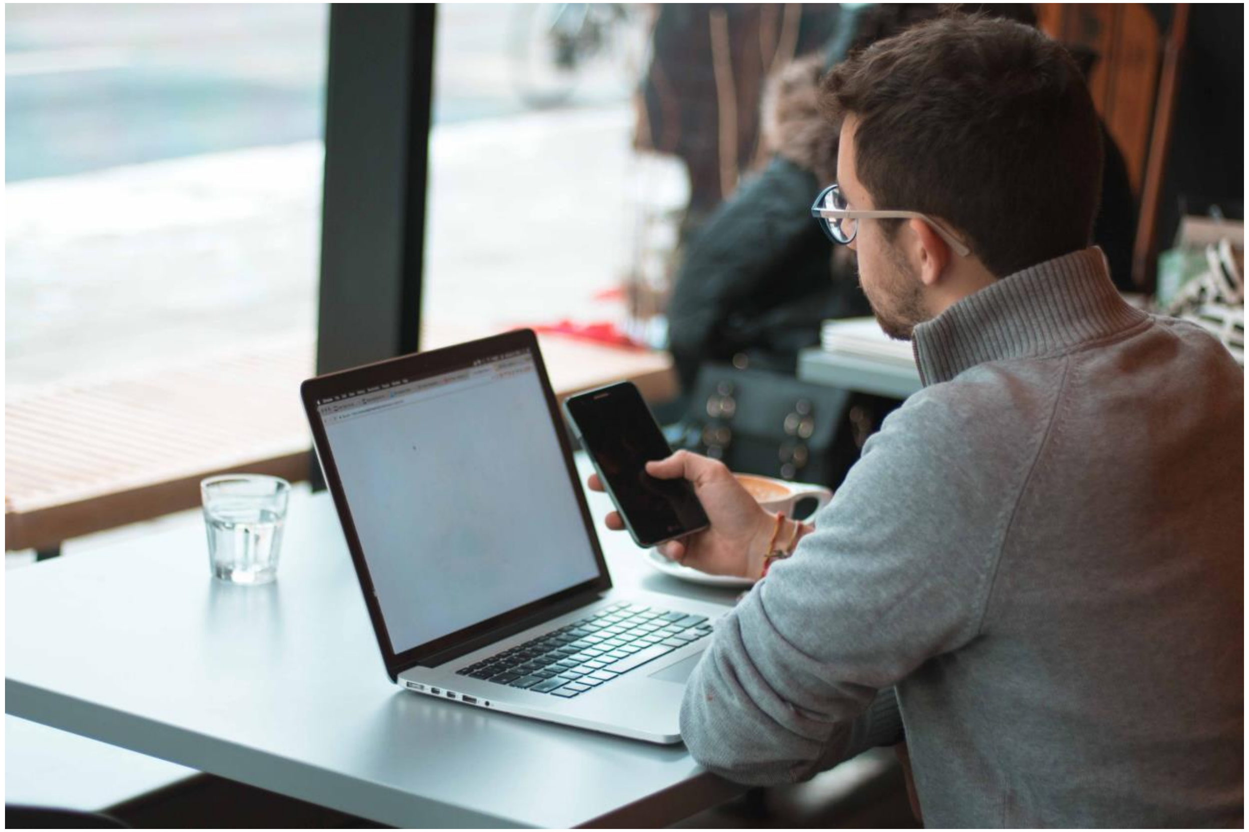
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

3. Select Time:

Friday, September 29, 2023

Time Zone: Eastern Time - US & Canada (change)



Man sitting near table with laptop photo by Joseph Frank on Unsplash

Top 10 resources you should know about!

1. Zoterobib
2. Zotero
3. Omni
4. Borrowing from Other Libraries
5. Brock Library NEUR Library Research Guide
6. Google Scholar, Google Books and Advanced Google searching
7. Open Education Resources (OERs)
8. PubMed / MEDLINE
9. When, where and how to get help!
10. Book a Consultation

One thing you’ve learned today?

Ian Gordon

Teaching & Learning Librarian
igordon@brocku.ca

All the best with your assignments, writing, conversations and learning.

Library Seminar Agenda

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- What is neuroscience?
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- Scholarly resources – databases
- Scholarly resources – articles
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